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GENERAL PRACTICE SERIES

THE PITUITARY GLAND IN DISEASE*

R. HOFFENBERG, M.D., M.R.C.P.

and

W. P. U. JACKSON, M.A., M.D., M.R.C.P.

From the Endocrine Clinic, Department of Medicine, Groote Schuur Hospital, and the University of Cape Town

It is not yet certain how many hormones are secreted by the pituitary gland. Among the more important secretions—from a clinical point-of-view—are:

1. *Adrenocorticotrophic Hormone (Corticotrophin, ACTH)*

This stimulates the adrenal cortex, but *not* the medulla.

2. *Thyrotrophic Hormone (Thyroid-stimulating Hormone, TSH)*

This promotes the formation of thyroid hormone from iodine by the thyroid gland, and its secretion into the circulation.

3. *Gonadotrophins*

These consist of: (i) Follicle-stimulating hormone (FSH) (ii) Luteinizing hormone (LH), and (iii) Prolactin (lactogenic hormone, luteotrophin, mammotrophin).

FSH stimulates ovarian follicular growth mainly; LH is chiefly responsible for oestrogen and progesterone secretion by the ovary; prolactin has an action auxiliary to that of LH in promoting function of the corpus luteum and is the chief hormone concerned with lactation.

These hormones have analagous effects in the male. Thus FSH stimulates spermatogenesis by the testicular tubules, whereas LH—known as interstitial-cell-stimulating hormone (ICSH) in males—promotes androgen secretion.

4. *Somatotrophin (Growth Hormone)*

This is important in the regulation of the rate of body growth and its ultimate size. Its effects are manifested throughout the body. It plays an important role in the metabolism of protein, fat, carbohydrate, water and minerals.

5. *Vasopressin (Antidiuretic Hormone, ADH)*

This hormone of the posterior pituitary gland is concerned primarily with water metabolism.

DISORDERS OF THE PITUITARY GLAND

Disorders of the pituitary gland may arise from:

1. *Non-functioning Tumours*

These cause 'neighbourhood' pressure effects particularly involving the optic chiasma or tracts, classically producing bitemporal hemianopsia.

2. *Hyperfunction*

The effects vary according to the type of hormone produced in excess:

(i) *ACTH* acting through the adrenal glands may produce Cushing's syndrome in some patients.

(ii) *TSH*—possibly responsible for some cases of thyrotoxicosis by stimulating the thyroid excessively.

(iii) *Growth hormone*—responsible for gigantism (if a growing individual is affected) or acromegaly (in a fully-grown adult). This is the only clear-cut example of pituitary-gland hyperfunction, neither of the other states being accompanied by enlargement of the pituitary fossa.

3. *Hypofunction*

This usually affects all the 'trophic' hormones. Pituitary deficiency states include:

(i) *Panhypopituitarism* (Simmond's disease, Sheehan's syndrome).

(ii) *Pituitary dwarfism*—resulting from the rare state of hypopituitarism in a growing child.

* This is the 3rd article in a series of 5 on endocrine disturbances by the same authors which are appearing weekly.

(iii) *Diabetes insipidus*—where the posterior pituitary gland is inactive.

SOME COMMON CLINICAL ERRORS

1. *Cushing's Syndrome*

Previously called 'pituitary basophilism', this condition was once uniformly attributed to a basophil adenoma of the pituitary gland. Present concepts incriminate the adrenal glands, although cases due to adrenal hyperplasia possibly are mediated by excessive ACTH production at some time. Therapy is directed, therefore, toward the adrenal glands in most cases. This condition is not common and will be considered in a subsequent article on the adrenal glands.

2. *Obesity*

Obesity always results from a calorie intake excessive for the metabolic needs of the patient. This is *not* the result of of 'glandular' dysfunction. In particular, recent thought has tended to cast discredit on the concept of 'Fröhlich's syndrome'. This designation should be reserved for patients with obesity and true hypopituitarism due to a recognizable hypothalamic lesion—a very rare state of affairs indeed. Obese children of the 'Fröhlich's' type need calorie restriction, *not* hormonal therapy.

3. *Pituitary Dwarfism*

This is another overworked diagnosis. When general metabolic disorders and bone dysplasias have been excluded, a small core of endocrine diseases remains as a cause of dwarfism. The majority of these appear to be primarily hypogonadal. Cases of dwarfism require specialized investigation.

4. *Panhypopituitarism*

Most of the confusion about this condition has arisen from the equation of Simmond's disease with 'pituitary cachexia'. It is realized today that the great majority of these patients do not present a cachectic picture. Cases of anorexia nervosa are often mistakenly diagnosed as having pituitary hypofunction. Extensive investigations may be required to distinguish these two conditions, as secondary endocrine-gland suppression frequently accompanies anorexia nervosa.

Clinical points which distinguish anorexia nervosa from the unusual thin patient with pituitary failure are:

(i) Despite considerable emaciation, patients with anorexia nervosa exhibit restlessness and often cheerfulness; hypopituitary cases are generally inert and apathetic.

(ii) Retention of sex hair is characteristic of anorexia nervosa; in addition these patients often show fine downy hair-growth over the face and back (as seen in other states of inanition). In hypopituitarism loss of sex hair is a cardinal feature.

In hypopituitarism, as a result of diminished TSH stimulation, the production of thyroid hormone is deficient. The majority of patients with hypopituitarism thus present a hypothyroid picture—so-called 'pituitary' or 'secondary myxoedema'. These patients are as a rule well-nourished or slightly overweight.

The commonest etiology is necrosis of the pituitary following postpartum haemorrhage or severe shock. These patients almost invariably do not lactate. Amenorrhoea, loss of libido and sex hair, and hypothyroid features, complete the diagnosis.

It is important to exclude a pituitary cause in cases of myxoedema, as treatment with thyroid alone is unlikely to succeed and may be hazardous. Hypopituitarism often presents a picture of insiduous decline in health with loss of libido and physical and mental torpor as its only features. Occasionally it is the cause of an obscure 'refractory' anaemia.

5. *Diabetes Insipidus*

This is a rare cause of polyuria and polydipsia. The basic defect is an inability to concentrate the urine, and polyuria is obligatory in order to rid the body of its excretory products. As a secondary effect polydipsia occurs. Other conditions which may simulate diabetes insipidus are:

(i) Psychogenic polydipsia—where the patients drink excessive amounts of fluid from habit or because they feel that it is good for them.

(ii) Renal disease.

(iii) Diabetes mellitus.

The second and third conditions are not difficult to distinguish. A simple test to exclude psychogenic polydipsia is a water-deprivation test. Patients with true diabetes insipidus will seldom tolerate this test and will not produce a concentrated urine. Psychogenic polyuria manifests itself by concentration of the urine—usually within 6-12 hours of water deprivation. An early-morning urinary specific gravity will often suffice to distinguish the two conditions. Specialized tests are available in difficult cases.

ABSTRACT : UITTREKSEL

Congenital Syphilis in the Absence of Positive Serology in the Mother. Nicholas J. Fiumara and William Hill. J. Amer. Med. Assoc., **163**, 1037. 23 March 1957.

A case is described of an 8-year-old boy with positive blood tests for syphilis, bulging frontal bosses, a saddle nose and Hutchinson's teeth suggesting congenital syphilis, whose mother showed no history or clinical evidence of syphilis, and whose standard blood tests were repeatedly negative. On prolonged and detailed examination of the mother over 6 months it was found that a few hours after the birth of the patient she had been found to have a temperature, headache, sore throat, and a maculopapular rash. A diagnosis of syphilis was not made but she was treated with penicillin. No blood test was taken. It was found that when the patient was 5 months old the mother was married a second time and a blood test the day after the ceremony was positive; 6 injections of Mapharsen were given. The doctor did not report the case or examine the husband. An interesting feature of this test is that the Treponema Immobilization Test gave positive results on the mother when Hinton and Kahn tests were negative at the time that the syphilis was diagnosed in the 8-year-old child. It is suggested that a new use for the T.P.I. test is to assist in the diagnosis of syphilis in a patient with epidemiologic evidence of the disease but with negative clinical and serologic findings. It is also suggested that the efficiency of modern treatment leads to careless diagnosis and the disease not being recognized.

F.W.F.P.

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EDITORIAL

NEW VIEWS ON AORTIC STENOSIS

Surgical treatment of the orifices and valves of the heart was a logical field for development as the result of the progress made in thoracic surgery during and after the last war. Of the 2 valves commonly afflicted (the mitral 5 times more frequently than the aortic) the aortic has always been the neglected step-child of the cardiac surgeons while mitral valvotomy has progressed by leaps and bounds to become a standard procedure in most large hospitals. Sir Russell Brock, of Guy's Hospital, London, one of the pioneers as well as the champion of aortic valvotomy, surveys the surgical treatment of aortic stenosis in a recent issue of the *British Medical Journal*.[1] His conclusions, based on his own series (120) and those of his British and American colleagues (about 320 cases), are emphatically favourable towards a wider application of surgical treatment of the condition. The operative difficulties and mortality, though fully discussed, are made light of, and Brock draws some vivid clinical analogies that may well indicate the eventual status of, and outlook upon, cardiac valvular stenosis in medicine. For example, he likens the state of obstruction of the left ventricle to obstruction in other bodily hollow muscular organs such as the colon and the bladder, and maintains that the same principles should be followed to overcome it. He emphasizes that 'one of the basic functions of the surgeon is to relieve obstruction to a hollow viscus or duct . . . The relief of obstruction to the oesophagus, the pylorus, the intestine, the urinary or biliary apparatus is accepted, without question, as a surgical matter . . . . Neglect of chronic or subacute obstruction of the bladder or the colon would be considered professional incompetence or neglect, and yet chronic or subacute obstruction of the left ventricle is still viewed with complacency'. According to Brock the operative mortality in an average-risk patient with isolated aortic stenosis should be no more than 5%; but when advanced secondary changes are present the operative risk may rise to 25 or even 50%

This aspect, then, is the crux of the matter: pre-operative assessment. Here Brock will undoubtedly arouse controversy with his unorthodox views upon the assessment of the gravity of aortic stenosis, which he introduces with the remark that 'there has been much loose and immature thought'

VAN DIE REDAKSIE

NUWE MENINGS OOR SLAGAARSTENOSE

Chirurgiese behandeling van die openinge en kleppe van die hart was 'n logiese terrein vir ontwikkeling as gevolg van die vooruitgang op die gebied van borschirurgie gedurende en na die afgelope oorlog. Van die 2 kleppe wat gewoonlik aangetas word (die myter-5-maal meer dikwels as die slagaarklep), was die slagaarklep altyd die verwaarloosde stiefkind van die hartchirurge terwyl mytervalvotomie met rasse skrede vooruitgegaan het en die standaard metode by die meeste groot hospitale geword het. In 'n onlangse uitgawe van die *British Medical Journal*[1] gee sir Russell Brock, van Guy's Hospitaal, Londen, een van die baanbrekers asook die voorvegter van slagaarvalvotomie, 'n oorsig oor die chirurgiese behandeling van slagaarstenose. Sy gevolgtrekkings, gebaseer op sy eie reeks (120) en dié van sy Britse en Amerikaanse kollegas (ongeveer 320 gevalle), is uitdruklik ten gunste daarvan dat die toestand meer algemeen chirurgies behandel moet word. Die operatiewe moeilikhede en sterftesyfer, alhoewel dit ten volle bespreek word, word as van weinig belang beskou, en Brock maak 'n paar lewendige ooreenstemmings wat moontlik die uiteindelike status van, en die vooruitsig op hartklepstenose in die geneeskunde mag aandui. Byvoorbeeld, hy vergelyk die toestand van verstopping van die linkerhartkamer met verstopping by ander hol liggaamlike spierorgane, soos bv. die dikderm en die blaas, en hou vol dat dieselfde beginsels gevolg behoort te word om dit te bowe te kom. Hy beklemtoon dat ‚een van die basiese funksies van die chirurg is om verstopping van 'n hol inwendige orgaan of buis te verlig . . . Die verligting van verstopping van die slukderm, die maaguitgang, die derm, die urien- of galstelsel word sonder twyfel aangeneem as 'n chirurgiese saak . . . Verontagsaming van chroniese of subakute verstopping van die blaas of die dikderm sou as professionele onbevoegdheid of nalatigheid beskou word, en tog word chroniese of subakute verstopping van die linkerhartkamer nog onbekommerd gadegeslaan'. Volgens Brock behoort die operatiewe mortaliteit by 'n gemiddelde-risiko pasiënt met afgesonderde slagaarstenose nie hoër as 5% te wees nie; maar wanneer daar gevorderde sekondêre veranderinge aanwesig is, mag die operatiewe risiko tot 25 of selfs 50% styg.

Hierdie aspek dus, is die kern van die saak: voor-operatiewe skatting. Hier sal Brock ongetwyfeld 'n polimiek aan die gang sit met sy onortodokse menings oor die skatting van die ernstigheid van slagaarstenose, wat hy inlui met die

about the subject. He believes, for instance, that there is no such entity as a mild case of aortic stenosis—they are all serious, or potentially so. The fact that, in a series of 78 cases, 2 reached the age of 60 should not be allowed to obscure the fate of the 31 who died before they were 40. If, then, we accept Brock's view on the seriousness of the condition we must know which cases are the most likely to benefit by surgical operation. In this respect aortic stenosis is a particularly difficult condition to adjudge, for initially there may be no symptoms or signs at all apparent to the patient or his medical attendant. Unlike cases of stenosis of the mitral valve, aortic stenotics show no sign of heart failure until the final stage. (Brock aptly likens the heart of a sufferer to the willing horse, which goes on until it can't go any further and then drops dead.) When signs of right or left heart failure supervene, Brock considers the case no longer operable. Brock asks, does the clinician wait until the man with a chronic prostatic obstruction has a bladder up to his umbilicus, his urinary tract infected and his kidneys barely functioning before turning him over to the surgeon for operation? 'Yet how often is this the very time that the patient (with aortic stenosis) is first directed to the surgeon.' The physician may defend his policy of not referring his patients on the ground that he feels that their disability does not warrant it. While this decision is undoubtedly valid in many cases, it is equally wrong in others, for it is an empiric one based on a clinical opinion that Brock describes as no more than a 'hunch'. His reasons for this are that the clinician's standards are faulty—either because they are too circumscribed or because they are irrelevant. He considers that the simplest assessment of all—that based on the patient's symptoms—is also the most ingenuous, because the need of an operation is hardly ruled out by the fact that the patient is unaware of any mischief—that is a decision for his doctor. The value of all symptoms and signs has to be carefully weighed, but Brock appears to rely largely upon cardiac catheterization, which he uses to find the difference in pressure between the blood in the left ventricle and that in the aorta and peripheral vessels. A simple, quick—and probably the safest—way of determining the ventricular pressure is by percutaneous puncture whilst the pressure on the aortic side of the valve is determined at the femoral or brachial artery. Two cases might exhibit identical clinical pictures yet have widely differing pressure gradients. Brock states that in general a gradient of 50 mm. Hg or over is an indication for operation, irrespective of the clinical picture. It is clear that Brock lays at least as much store upon this objective unit of assessment as upon the general clinical examination. It may well be that he is right, that success of the operation depends on the differences of pressure rather than upon the severity of the clinical symptomatology. While Sir Russell Brock's provocative essay will not find general acceptance, his brilliantly-presented thesis will be generally studied around the world.

1. Brock, R. (1957): Brit. Med. J., **1**, 1019.

opmerking dat ‚daar baie losse en onrype idees' oor die onderwerp was. Hy glo bv. dat daar nie so iets soos 'n ligte geval van slagaarstenose bestaan nie—hulle is almal ernstig of potensieel so. Die feit dat uit 'n reeks van 78 gevalle 2 die ouderdom van 60 bereik het, behoort nie toeglaat te word om die lot van die 31 wat gesterf het voor hulle 40 was, in die skadu te stel nie. As ons dus Brock se mening oor die ernstigheid van die toestand aanneem, moet ons weet watter gevalle die grootste kans sal hê om by chirurgiese behandeling te baat. In hierdie verband is slagaarstenose 'n besonder moeilike toestand om oor te beslis, want aanvanklik mag daar glad geen simptome of tekens vir die pasiënt of sy geneesheer sigbaar wees nie. Anders as gevalle met stenose van die myterklep, toon lyers aan slagaarstenose geen tekens van hartondoeltreffendheid tot by die uiteindelike stadium nie. (Heel gepas vergelyk Brock die hart van 'n lyer met die gewillige perd wat aanhou tot dit nie verder kan gaan nie, en dan dood neerslaan). Wanneer tekens van regter- of linker-hartondoeltreffendheid tussenbeikom, beskou Brock die geval nie langer as opereerbaar nie. Brock vra, wag die klinikus totdat die blaas van die persoon met 'n chroniese prostaatverstopping tot by sy nawel is, sy urienweë besmet en sy niere skaars werk voordat hy hom vir 'n operasie na die chirurg stuur? ‚En tog, hoe dikwels is dit nie presies dan eers dat die pasiënt (met slagaarstenose) vir die eerste keer na die chirurg gestuur word nie.' Die internis mag sy beleid, om nie sy pasiënte na die chirurg te verwys nie, verdedig op grond van die feit dat hy voel dat hul ongesteldheid dit nie regverdig nie. Terwyl hierdie besluit ongetwyfeld by baie gevalle gegrond is, is dit net so verkeerd by andere, want dit is 'n empiriese besluit gebaseer op 'n kliniese mening, wat Brock as niks meer as 'n ‚voorgevoel' beskryf nie. Sy redes hiervoor is dat die klinikus se standaarde gebrekkig is—òf omdat hulle te afgebaken is, òf omdat hulle nie toepaslik is nie. Hy reken dat die eenvoudigste skatting van almal—dié gebaseer op die pasiënt se simptome—ook die onskuldigste is, aangesien die nodigheid van 'n operasie skaars uitgeskakel word deur die feit dat die pasiënt onbewus is van enige moeilikheid—dit is 'n besluit vir sy dokter. Die betekenis van alle simptome en tekens moet sorgvuldig oorweeg word, maar dit skyn of Brock tot 'n groot mate op hart-kateterisering staatmaak, wat hy gebruik om die verskil in drukking tussen die bloed in die linkerhartkamer en dié in die slagaar- en perifêre vate te vind. 'n Eenvoudige, vinnige—en waarskynlik die veiligste—wyse om die hartkamerdrukking vas te stel, is deur 'n perkutaanse prikgaatjie, terwyl die drukking aan die slagaarkant van die klep by die dybeen- of bo-armslagaar vasgestel word. Twee gevalle mag identiese kliniese beelde bied, en tog totaal verskillende drukkingsgradiënte hê. Brock verklaar dat 'n gradiënt van 50 mm. Hg of meer oor die algemeen 'n aanduiding vir operasie is, ongeag van die kliniese beeld. Dit is duidelik dat Brock ten minste net soveel waarde aan hierdie objektiewe eenheid van skatting as aan die algemene kliniese ondersoek heg. Dit mag wel wees dat hy reg is dat die sukses van die operasie afhang van die verskille in drukking eerder as van die ernstigheid van die kliniese simptoomleer. Terwyl sir Russell Brock se prikkelende verhandeling nie algemeen aangeneem sal word nie, sal sy skitterend-aangebode tesis algemeen deur die wêreld bestudeer word.

1. Brock, R. (1957): Brit. Med. J., **1**, 1019.

THE BACTERIAL WALL

In the last few years it has become possible to identify many of the constituents of the bacterial wall.[1] The investigation of the chemical composition of the cell walls, hitherto unknown, has been made possible by special technical advances which allow relatively homogenous walls to be obtained from mechanically disrupted cells. The cell walls are prepared as visible collapsed structures free from cytoplasm, which is removed by washing and differential centrifugation, or by enzymes. The walls contain lipids, phosphorus, hexosamines, amino acids, and usually carbohydrates, but no nucleic acids, purines, or pyrimidines. The commonest sugars are glucose, mannose and galactose. Glucosamine is always present, and another hexosamine called 'muramic acid'. Glutamic acid and alanine are the amino acids always present, but others occur in varying numbers. There is a difference in the composition of the cell walls from Gram-positive and Gram-negative bacteria, the latter group containing, for example, 10 times more fat and 10 times less hexosamine than Gram-positive organisms; their amino-acid composition is also more complex. It would appear that 'each bacterial genus may have a distinctive pattern of cell-wall components in particular among the amino acids present.'

The cell wall is apparently not essential for the life of the bacterial cell and when removed by enzyme digestion the remaining fragile body can continue to synthesize proteins and nucleic acids, grow and divide, provided it is protected from osmotic and mechanical stresses. The composition of the components of the interior of bacteria differs from that of the cell wall.

On the other hand, the action of penicillin on bacteria, according to another view, is that it inhibits the synthesis of bacterial cell wall; the morphological changes it produced in bacteria (the large forms) may be due to extrusion of the unstable 'protoplast' through points where new wall formation has not occurred. Much work has been done with a view to determining the enzyme system affected by penicillin and its site of action in the cell.

Many enzymes, such as lysozyme, attack cell walls and cause lysis of bacteria. Many species of bacteria produce enzymes that can lyse cell walls and, as with lysozyme, the sensitivity to digestion varies a good deal; some species may undergo complete dissolution, others show complete resistance. The fractions obtained from the enzymic digestion of cell walls have been investigated. In general it would appear that the cell walls of Gram-positive bacteria consist of an insoluble basal structure of chains of acetylated glucosamine and muramic acid, bound to peptides, which may be highly cross-linked or cyclic; polysaccharides and phosphorylated compounds may also be attached. It is also known that the constitution of the walls differs in different species.

The subject is naturally a very complex one; details of the biochemistry may be found elsewhere.

1. Work, E. (1957): Nature, **179**, 841.

INFLUENZA, A GENERAL REVIEW OF RECENT DEVELOPMENTS*

JAMES GEAR

and

M. A. WESTWOOD

WHO Influenza Centre for Southern Africa, South African Institute for Medical Research, Johannesburg

Influenza is a febrile illness of Man caused by a virus, *Myxovirus influenzae*, of which there are at least 3 distinct immunological types. This virus has a predilection for the epithelium of the respiratory tract. The infected cells undergo necrosis, which is followed by inflammation involving the various parts of the tract in a varying degree in different cases. The resulting illness is characterized by a sudden onset, headache, prostration, and fever, and symptoms and signs of involvement of the respiratory tract, including coryza, sore throat, slight cough, and in some cases pneumonitis. The death rate of uncomplicated influenza is very low, but pneumonia complicating influenza is one of the commonest causes of death, especially of the old and frail.

RECENT HISTORY

The cause of the influenza epidemics of the late 19th century and of the 1918-19 pandemic was not ascertained. The influenza virus was first isolated in 1933 by Wilson Smith, Andrewes, and Laidlaw[1]. They transferred the infection from human cases by instilling their throat washings intranasally into ferrets, which then developed a febrile condition associated with coryza and sneezing. The first strain isolated was passed serially from ferret to ferret. On recovery the ferret was immune to challenge with the same virus, which was also neutralized by sera from convalescent patients. Later the virus was transmitted from ferrets to white mice, which greatly simplified its study. This was still further facilitated by its adaption to embryonated eggs[2] and the subsequent finding that it caused red cells to agglutinate.[3] This property of causing haemagglutination has been of the greatest value in the study of the influenza virus and has had many important practical applications.

A second immunologically distinct type of virus was isolated independently in 1940 by Francis and by Magill,[4] from throat washings of patients with influenza. This virus was thus called Influenza B, and the type isolated previously, Influenza A.

* Written and published at the request of the Secretary for Health.

A third immunologically distinct type was isolated by Taylor in 1947,[5] and a similar strain was isolated in 1949 by Francis, Quilligan and Minuse[6] from cases of illness indistinguishable from mild influenza. This virus has been named Influenza C.

In 1952 Kuroya N. *et al.*[7] investigated an outbreak of pneumonitis among newborn infants in a hospital at Sendai in Japan. The onset was sudden, with fever, cyanosis, and marked dyspnoea; 12 of 17 patients died. From 5 of the fatal cases a virus was isolated by the intranasal instillation of mice with lung extracts. It was adapted to grow in embryonated eggs and was later found to cause haemagglutination of fowl red cells. It is now known as the Sendai virus. Its characteristics place it in the same group as the influenza viruses. Evidence of infection by the Sendai virus has been found in the United States[8] and more recently in Britain.[9] The illnesses in the patients studied varied from a mild influenza-like illness to severe pneumonia.

In studies on the aetiology of cases of acute laryngo-tracheo-bronchitis in Toronto in 1953-54, Morgan *et al.*[10] found that there was no evidence to incriminate a specific bacterial cause in the majority of cases, but from 5 cases an agent, producing cytopathogenic changes in tissue culture of human embryonic lung and monkey kidney cells, was isolated.

More recently Chanock[11] has described the isolation of a virus from cases of infantile croup in Cincinnati. The infants from whom this virus was isolated, as well as some others with the same condition, developed significant increases in neutralizing antibody, or haemagglutination-inhibiting and complement-fixing antibody, during convalescence—findings clearly suggesting that this virus was responsible for their illness. Its size has been estimated to be 90-135mμ in diameter. It was not ether-resistant and it agglutinated chick red cells and human O red cells. These properties are those required for placing it in the myxovirus group.

The Toronto virus was found to have similar properties and to be indistinguishable by haemagglutination-inhibition tests from one of the croup-associated viruses isolated in Cincinnati. No antigenic relationship with influenza virus A, A^1, B or C, Newcastle-disease virus or Sendai virus was found. It appears, then, that another immunologically distinct virus of this group has been uncovered. Thus, up to the present, the following influenza viruses or influenza-like viruses have been recognized:

Myxovirus influenzae		A
,,	,,	A^1
,,	,,	B
,,	,,	C
,,	,,	D (Sendai virus)
,,	,,	E (The virus associated with infant croup in Toronto and Cincinnati)

THE VIRUSES OF INFLUENZA

These viruses are all members of the Myxovirus group, which also includes, among others, the virus of mumps and the virus of Newcastle disease of fowls. The viruses of this group are characterized by being moderately large viruses, occurring as spheres varying from 80 to 150 mμ in diameter, or as filaments of about the same diameter and up to several microns in length. They are stable at —76°C but less so at —10°C, and their infectivity is readily destroyed by treatment with 20% ethyl ether. They also have the property of causing red cells to agglutinate, apparently resulting from adsorption of the virus onto receptors on the surface of the red cell. Haemagglutination and its inhibition by the corresponding specific antiserum has been extensively used in the study of this group of viruses, particularly of their immunological relationships.

These studies have shown that the influenza A viruses have been undergoing a progressive series of antigenic changes with the frequent appearance of new varieties. The WS subtype was found in 1933. From 1934 to 1945 the strains were of the PR8 subtype. The strains isolated from 1946 and subsequently have been so different antigenically from the A strains isolated earlier that they have been allotted to a new subtype, the A prime (A^1) subtype. Strains of this subtype isolated in one epidemic tend to be serologically uniform, but those isolated in different epidemic years show definable differences. All influenza A viruses share a common soluble antigen which may be demonstrated by the complement-fixation test.

There is also evidence that the influenza B viruses include a number of different subtypes, but generally the influenza B Strains are more uniform serologically than the A viruses.

The antigenic variations within the influenza C type, the Sendai type, and the myxovirus associated with infantile croup, have not yet been defined.

'*Asian*' *Influenza*. Viruses having the characteristics of the Myxovirus group have been isolated from cases of 'Asian' influenza occurring in Hong Kong, Malaya, the Phillipines, India and, more recently in Holland and the United States of America. These viruses are apparently immunologically similar, and by complement-fixation tests have been shown to be strains of influenza A type. However, the haemagglutination-inhibition tests have revealed that these strains are antigenically distinct from all previously isolated strains of this type. Preliminary studies of the sera of persons of different ages carried out in Holland suggest that many people over the age of 70 have antibodies against this virus, but almost none under this age have immunity. This interesting finding needs further investigation but, if confirmed, it suggests that the virus responsible for this epidemic is immunoligally related to the viruses responsible for the pandemic of 1890-91.

PATHOGENESIS

The influenza viruses have a predilection for the epithelium of the respiratory tract. The adsorption onto these cells is mediated through receptors on the surface of the cell. The virus multiplies within the cells and brings about their destruction. Large areas of the epithelial lining may thus be damaged.

CLINICAL PICTURE

The incubation period is 1-4 days, usually 1-2 days. The onset is characteristically sudden, with headache, muscle aches, and shivering. The illness is characterized by headache, anorexia, prostration, drowsiness, and muscle aching, especially of the limbs and the small of the back. The temperature rapidly rises to its maximum and the fever on the average lasts 3 days, but in many cases is shorter.

In the early stage of the illness the constitutional signs and symptoms are outstanding; later those arising from involvement of the respiratory tract become more prominent.

The nose becomes blocked and there is usually coryza and cough, but little sputum is produced. The throat is sore and dry, and the voice is usually changed, but marked hoarseness is not often noted. The appetite is decreased and there may be slight nausea, but vomiting and diarrhoea are not a feature of the illness.

On examination the patient has a flushed, weary face, and his eyes are congested. The throat may show congestion and enlargement of the tonsils and adenoids. The chest may be clear, but a few rales and rhonchi may develop, especially in the later stages of the illness. During the fever the pulse rate is increased in proportion to the temperature. The spleen and liver are not enlarged or tender and there are no other abnormalities of the abdomen.

In convalescence some patients complain of debility and depression, and often a bradycardia is found, but most cases recover rapidly and completely.

Complications are common in influenza and most often involve the respiratory system. Bronchitis and bronchiolitis occur frequently, and influenzal pneumonia is not uncommon. The occurrence of these complications is suggested by a prolongation of the fever, and they are revealed by the characteristic signs on examination of the chest.

In most epidemics an occasional case of an acute fulminating pneumonia is seen. In the 1918-19 pandemic such cases were common. Early in the illness the patient developed dyspnoea associated with sweating and tachycardia, followed soon by cyanosis, which rapidly deepened as the difficulty in breathing increased. Many such cases ended fatally within 24-48 hours of the apparent onset. This fulminating type of influenzal pneumonia may be caused by the action of the virus alone.

More commonly pneumonia complicating influenza is due to a secondary bacterial infection with pneumococci, streptococci, staphylococci, or *Haemophilus influenzae*.

DIFFERENTIAL DIAGNOSIS

Influenza has to be differentiated from a number of acute febrile illnesses, especially those involving the respiratory tract. Amongst the latter are particularly those infections caused by the recently discovered group of Adenoviruses. Often the differentiation of influenza, which may present as a mild coryza or as a rapidly fatal fulminating pneumonia, from a number of other infections which may present in similar fashion, is impossible on clinical grounds alone.

The features favouring a diagnosis of influenza are: its occurrence in epidemic form; its sudden onset, with at first a predominance of constitutional symptoms over respiratory symptoms; and fever which unless complicated by secondary bacterial infections is of short duration. In the absence of such complications a feverish illness lasting longer than 1 week is not likely to be influenza, and other causes of the illness should be sought.

LABORATORY DIAGNOSIS

The clinical picture of a fully developed case of influenza is fairly characteristic, but there are many gradations in its severity, and in the mild, less typical, cases, as in the severe fulminating cases, it may be necessary to appeal to the laboratory for help in the diagnosis.

For the isolation of influenza virus from a suspected case, the patient should be asked to gargle with nutrient broth for about 30 seconds. These throat washings should then be sent under refrigeration to the laboratory, by putting the bottle in a watertight plastic bag and wrapping well in cotton wool before placing in a thermos flask containing ice. At the same time a specimen of blood—the acute-phase specimen—should be collected and also sent to the laboratory. About 2 weeks later a second specimen of blood—the convalescent-phase blood—should be collected. The sera separated from these two specimens are tested for the presence of, and the titre of antibodies against, known influenza viruses, and also against the patient's own strain.

Isolation of the virus may be attempted by instilling ferrets intranasally with a suspension prepared from the patient's throat washings. If infected, the ferrets will develop a febrile illness associated with coryza. This may be apparent on the original inoculation. More often several ferret-to-ferret passages are necessary before the characteristic picture is developed.

Today the usual method of isolating a strain of virus is to inoculate a suspension of throat washings from the patient into the amniotic cavity of chick embryos. After one or two passages it is adapted to grow in the allantoic cavity. Its serological relationships can then be studied by the application of the haemagglutination technique. The influenza virus causes human O, guinea-pig, and chick red cells to agglutinate. The inhibition of this haemagglutination by specific antisera enables the type and subtype and strain of the virus to be identified.

The antibody contents of the acute- and convalescent-phase sera are measured by the complement- fixation test, or more commonly by the haemagglutination-inhibition test. A fourfold rise in the titre is significant and indicative of an infection by the corresponding virus.

TREATMENT

A patient with influenza should stay at home in bed. If possible he should be isolated in his own room separate from the rest of the family to diminish the risk of passing on a heavy infection. It is particularly important to ensure this for the protection of very young infants and the old and frail. The patient should be given a light diet with plenty of liquid, including fresh fruit drinks.

No drug or antibiotic at present available is of specific value in controlling the influenza virus infection. They should be reserved for the treatment of complications, in which, of course, their use is clearly indicated and may be life-saving.

PREVENTION

It is almost impossible to prevent the spread of influenza. In pandemics the only communities which have escaped have been those living on isolated islands. Much, however, can be done to minimize the severity of the illness by insisting that patients should stay at home and that they should not for any reason become over-fatigued, or over-stressed in other ways, during the illness.

Vaccines which have been shown to be effective have been produced. Those in general use are of the killed or inactivated type. The virus suspensions from which these vaccines are prepared are harvested from chick embryos. The method used in preparing these vaccines is briefly as

follows: Hen eggs are incubated for 10-12 days. They are then candled. The infertile and dead eggs are discarded. The live eggs are inoculated into the allantoic cavity with the virus through a hole punched in the blunt end. After this hole has been sealed, the eggs are re-incubated for 2 days. They are then candled again. The dead ones are discarded. The live eggs are then opened at the blunt end by burning round with a fine oxyacetylene flame. The shell membrane is lifted off. The allantoic membrane is torn and the vessels allowed to bleed into the allantoic fluid, which is then harvested. This fluid contains high titre of influenza virus, but it is further concentrated and purified either by spinning the virus down in a Sharples centrifuge or by allowing the red cells to settle in the cold, and then taking off the supernatant fluid and adding 1/10th its volume of saline. The red cells are then re-suspended by shaking gently and then the suspension is incubated for 2-3 hours at 37°C. During this incubation the virus elutes from the red cells, which can then be separated by centrifuging. The supernatant fluid, now containing virus in high concentration, is inactivated by the addition of formalin in appropriate concentration.

The vaccine so prepared is controlled bacteriologically to ensure its sterility. Its potency is determined by the inoculation of animals, usually mice, guinea-pigs or chickens, with 2 doses of vaccine with an interval of 14 days between inoculations. The animals are bled 1 week later and the haemagglutination-inhibition antibody content is determined. There should be a significant rise in titre, at least fourfold, in the post-inoculation specimen as compared with the pre-inoculation specimen.

In human trials vaccines so prepared have been shown to be effective in stimulating significant rises in antibody titre in the majority of individuals inoculated. In field trials it has also been found that, provided the vaccine contains the strain responsible for the current epidemic, it confers a significant amount of protection on those inoculated. This has emerged particularly from studies in schoolchildren in England. In one such trial there were 10 times as many cases in the unvaccinated children as in the adequately vaccinated children.

Unfortunately vaccines prepared from strains isolated in previous outbreaks are usually of much less protective value. To have maximum efficacy the vaccine must be prepared from the strain responsible for the current epidemic. To make this possible is one of the objectives of the World Influenza Centre set up in London by the World Health Organization. Regional Centres have been established at strategic centres in various parts of the world. In April 1957 an extensive outbreak of influenza began in Hong Kong. This epidemic then spread to other countries in the Far East and from there has spread widely in the Northern Hemisphere. Virus isolated in Malaya was identified as belonging to the A type by complement fixation, but was found to be different from the recent strains of previous epidemics. This finding was confirmed by the London Centre. Ampoules of virus were then flown by airmail to several centres, including institutions engaged in the preparation of influenza vaccine. Although it will be possible in many of these countries to prepare vaccine before the epidemic begins, unfortunately the amount that can be prepared is very limited. Reliance can therefore not be placed on prophylactic vaccination to prevent the spread of influenza in a country. Its use will be limited to the inoculation of key personnel so as to reduce the disorganization of a community which may result from an extensive epidemic of influenza.

Two inoculations of vaccine at an interval of 6 weeks or longer are recommended. In most instances, therefore, it may be possible to give only one before the epidemic begins. However, even one injection, particularly of patients who have had previous experience of influenza, may be of value in stimulating a certain degree of immunity.

As it will not be possible to prevent the spread of influenza by vaccination, the authorities concerned have to make arrangements to ensure that essential services are kept going, and that special arrangements which may be necessary are made to provide extra hospital accommodation to treat those cases whose condition requires hospital treatment. It is to be emphasized that uncomplicated cases of influenza should be treated at home. It is not necessary to send them to hospital. In providing such services the help of voluntary services such as the Noodhulpliga, the Red Cross, and St. John Ambulance Association will be essential.

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INFLUENZA PROVISIONS IN WESTERN PROVINCE

At a meeting held in Cape Town on 22 July an influenza liaison committee was established consisting of Dr. R. J. Smit (chief regional health officer, Union Health Department, Cape Town), Dr. D. A. van Binnendyk and Dr. J. G. Burger (director and deputy director, provincial hospital services), Drs. E. D. Cooper, J. P. de Villiers, F. C. R. Smit, J. W. Dreyer and A. N. Wilson (medical officers of health, Cape Town, Cape Division, Somerset West, Paarl and Worcester), Dr. L. Celliers (chief medical inspector, Education Department), Dr. M. Bailey (Cape Western Branch of the Medical Association), Dr. L. Been (Union Health Department), Mr. A. D. Forsyth Thompson (liaison committee of Red Cross, St. John and Noodhulpliga, and Mr. P. N. P. Saayman (senior health inspector, Cape Western section, South African Railways).

In a circular letter that is being sent to all local authorities in the Region it is stated that in the event of an outbreak of influenza about 20% of the beds in provincial hospitals would be made available by a temporary clearance. Dr. van Binnendyk announced that this provision could be made. Should this be insufficient, military and other hospitals would be called upon.

Each local authority in the Region would be asked to establish an action committee consisting of a councillor, the medical officer of health, the district surgeon, a representative of voluntary organizations, and the medical superintendent of the local hospital.

SOME IMPORTANT FEATURES IN THE DEVELOPMENT, STRUCTURE AND FUNCTION OF THE PAROTID SALIVARY GLANDS

D. J. DU PLESSIS, M.CH. (RAND), F.R.C.S. (ENG.)

Department of Surgery, Groote Schuur Hospital, Cape Town, and University of Cape Town

During a study of the disorders affecting the parotid salivary glands it soon became apparent that the manifestations of disease in this situation are largely controlled by developmental, structural and functional factors and that an understanding of these was essential for a correct appreciation of pathology and treatment. In this paper some features of outstanding importance are discussed in view of their relation to pathological manifestations in these glands.

DEVELOPMENT AND DEVELOPMENTAL DEFECTS

Embryology

The parotid salivary gland arises as an epithelial outgrowth from the ectodermal epithelium of the lateral prolongation of the primitive mouth at about the 6th week of intra-uterine life.[25, 39] It is thus not surprising to find that the parotid gland in many ways resembles other glands with a similar origin, e.g. breast, sweat and sebaceous glands, and that in 28% of normal parotid glands sebaceous glands may actually arise from the finer branches of the parotid duct[19, 33] as can be seen in Fig. 1. This latent potentiality of differentiation in adult duct epithelium is further illustrated by the presence of squamous epithelium,[12, 20, 29] epithelial pearls[13] and basal cells[12, 13, 20] in mixed salivary tumours and is in conformity with the view, previously expressed, that mixed salivary tumours may be regarded as specialized basal-cell carcinomata[13] with a close resemblance to certain basal-cell tumours of skin[12, 20, 21] and adamantinomata.[21, 48] The presence of these sebaceous glands also explains the occasional sebaceous adenoma found in the parotid salivary gland[16] and the much commoner muco-epidermoid tumours[16] and sebaceous carcinomata, of which 1 example has been encountered (Fig. 2). These sebaceous glands are much commoner in glands with parotid tumours,[34] rising to an incidence of 73% in glands containing muco-epidermoid tumours,[34] suggesting that epithelium with this latent potentiality to differentiate is more prone to give rise to tumours of ectodermal origin. These sebaceous glands are also much more frequently encountered in the parotid gland than in the submandibular salivary gland,[34] and one wonders whether this is an explanation for the higher incidence of mixed salivary tumours in the parotid gland.

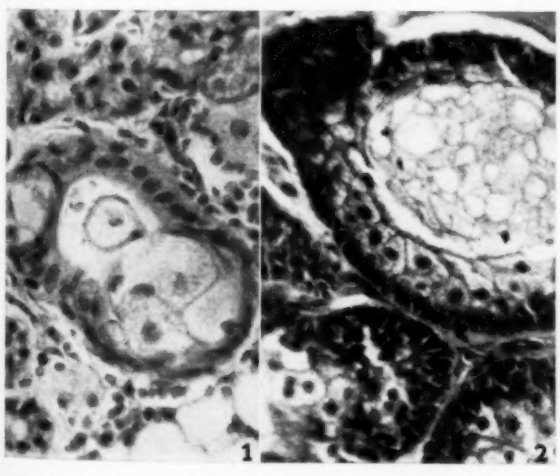

Fig. 1. Sebaceous glands arising from parotid duct.
Fig. 2. Sebaceous carcinoma in the parotid gland.

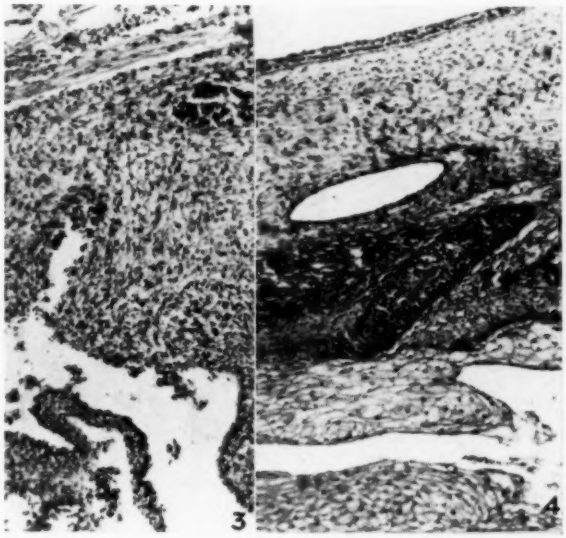

Fig. 3. 26 mm. embryo: normal development of parotid duct with formation of lumen.
Fig. 4. 47 mm. embryo, showing parotid duct arising from buccal cavity.

At first this outgrowth is a narrow cord of cells which soon develops a lumen[50] and this is illustrated by photomicrographs taken of a 26 mm. human embryo (Fig. 3) and a 47 mm. embryo (Fig. 4). This outgrowth elongates and extends backwards to just in front of and below the ear and at 47 mm. it has already started to branch at its termination (Fig. 8). In the 89 mm. embyro the gland lies chiefly in front of the ear (Fig. 5) but at 91 mm. it fills in the hollow below the ear (Fig. 6) and at this stage it consists of a freely branching system of ducts (Fig. 9). As the gland now enlarges it becomes wedged in between the mandible and the upper portion of the sternomastoid muscle as can be seen in Fig. 7 of a 121 mm. embryo. In the 138 mm. embryo the gland can be seen to have assumed a lobular structure with primitive acini, arising from the terminations of the finer ductules and lying together in little groups embedded in the surround-

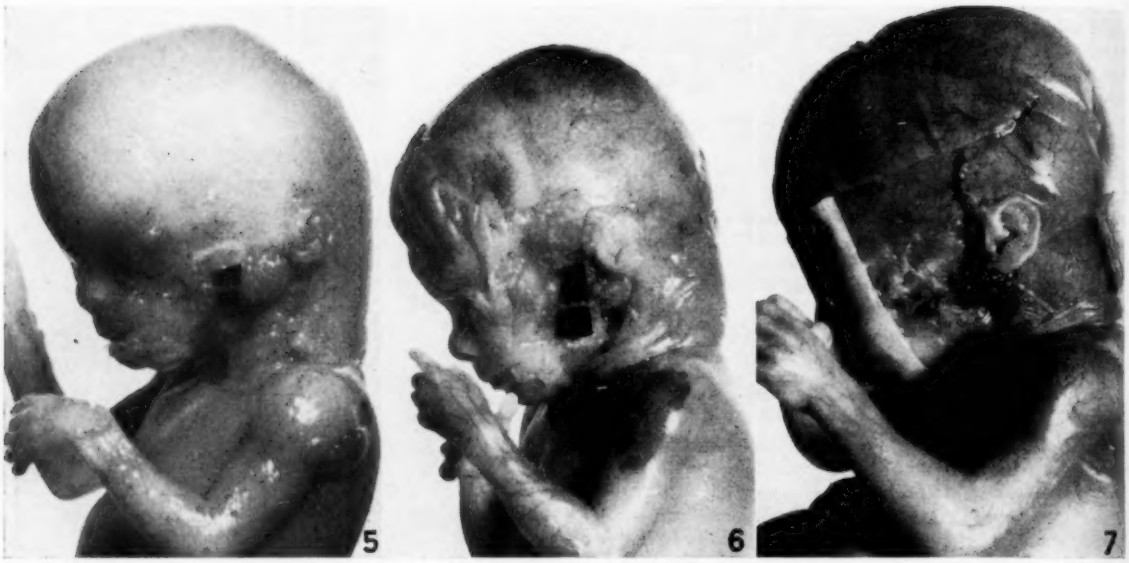

Fig. 5. 89 mm. embryo, showing dissection of parotid duct, with gland anterior to ear.
Fig. 6. 91 mm. embryo, showing dissection of parotid duct, with gland below the ear.
Fig. 7. 121 mm. embryo, showing parotid wedged in between mandible and upper part of sternomastoid.

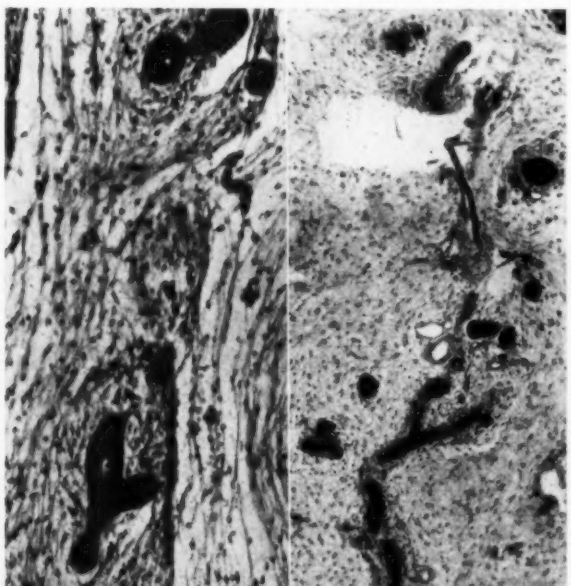

Fig. 8. 47 mm. embryo, showing branches of parotid duct.
Fig. 9. 91 mm. embryo, showing branching system of ducts in parotid.

ing mesoderm which will eventually form the capsule and the gland trabeculae (Fig. 11).

As the gland continues to enlarge it sends a deep prolongation between the two main branches of the underlying facial nerve to form a deep lobe of the gland—this can first be noticed in the 145 mm. embryo (Fig. 10). The gland becomes indented by the branches of the facial nerve; the classical relationship of these structures was described by McWhorter in 1917 with the branches of the nerve lying between the deep and superficial lobes of the gland.[28, 31]

Fig. 10. 145 mm. embryo, showing development of a deep prolongation of parotid between the 2 main branches of the facial nerve.

In many instances, however, the nerves so indent either one or the other of the lobes as to appear to run through the glandular tissue itself. This has been observed on many occasions and explains the report by Hurford that the nerve remained between the 2 lobes in 4 out of 11 cases only.[23] This will also explain the occurrence of permanent facial paralysis after superficial lobectomy of the parotid gland and it follows that during this operation the facial nerve and its branches must be dissected out under vision.

Accessory Parotid Gland

During this development a branch may grow out from the main duct itself (and not from its termination, as is the normal course of events) to produce a replica in miniature of the parotid gland—an accessory parotid gland. It is said to be present in 12% of glands[1] but it is probably

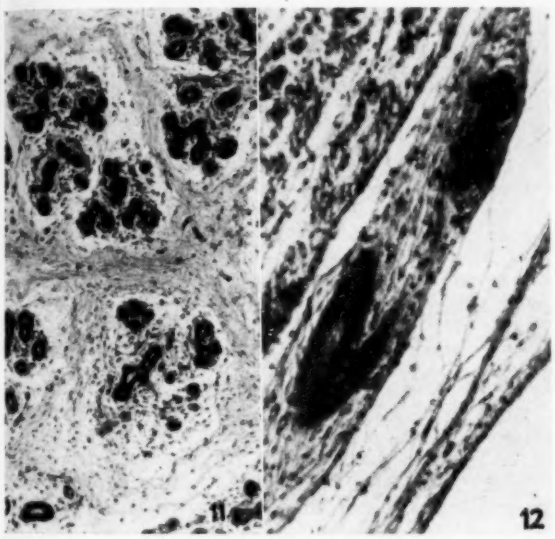

Fig. 11. 138 mm. embryo, showing gland with primitive acini.
Fig. 12. 47 mm. embryo, showing accessory gland arising as an outgrowth from main duct.

commoner than that, having been found in 3 out of 9 embryos that I have examined and in 33 out of 123 sialograms studied, i.e. 26·8%. This accessory gland arises as an outgrowth from the main duct and Fig. 12 shows it in a 47 mm. human embryo. In the adult it lies in the soft tissue of the cheek above the main duct, as can be seen on the sialogram in Fig. 13. This abnormality may be unilateral or bilateral and, of 19 people with bilateral sialograms, 4 showed accessory glands on both sides and 4 showed it on one side only.

Such an accessory parotid gland is naturally heir to all the diseases of the main gland, in which case the lesion will present in the cheek and the diagnosis may easily be missed if this abnormality is not kept in mind. Fig. 14 shows a sialogram with sialectasis in the accessory parotid gland and Fig. 15 shows a sialogram of a patient with a carcinoma of the accessory parotid gland displacing the duct downwards.

Congenital Stenosis

The normal development may be disturbed in many ways to produce congenital abnormalities of the gland or its duct.

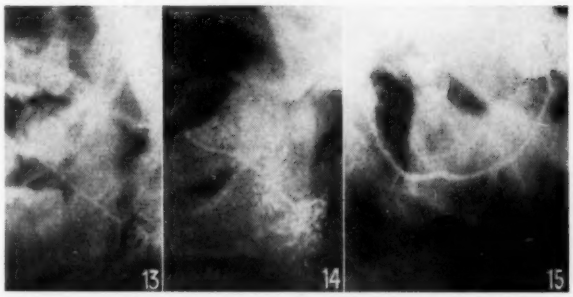

Fig. 13. Sialogram in adult, showing accessory duct.
Fig. 14. Sialogram showing sialectasis in accessory parotid gland.
Fig. 15. Sialogram showing displacement of parotid duct by carcinoma of accessory parotid gland.

An occasional finding is congenital stenosis of the parotid duct orifice which will produce obstructive symptoms. This condition is rare and must only be diagnosed after excluding the commoner traumatic and inflammatory causes for the stenosis. On this basis one has seen this condition only twice, in one case stenosis was present at all 4 orifices of the major ducts.

Congenital Displacement

An exceptionally rare developmental abnormality has been seen in one patient who had a soft-tissue defect on one side of his face (Figs. 16 and 17) resulting in failure of the parotid gland to migrate to its normal situation, and sialograms revealed it to be higher and further forward than normal.

Congenital Sialectasis

Congenital sialectasis is an interesting condition. It has been held to be a developmental disturbance[3, 27, 38] with the production of dilated alveoli at the terminations of the finer ductules: this is said to produce stasis and so to predispose to recurrent parotitis.[3, 27, 38, 46, 51] There is no embryo-

Figs. 16 *and* 17, showing primitive abnormal situation of parotid gland.

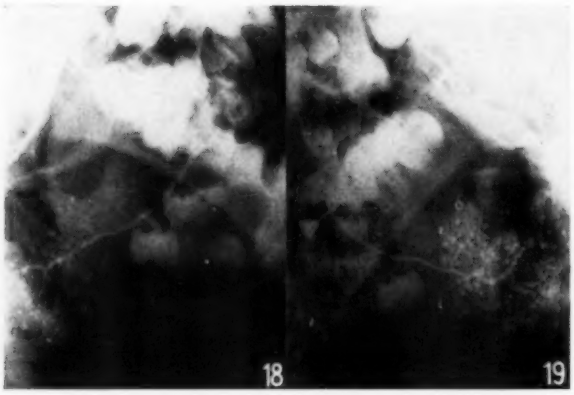

Fig. 18. Sialogram showing sialectasis after one attack of parotitis.
Fig. 19. Sialogram showing sialectasis in a boy of 9 after 5 attacks of parotitis.

logical explanation for such a defect and one hesitates to accept the condition as an entity. Sialectasis is unquestionably seen on sialography in very young children, but these glands have always been the site of previous attacks of parotitis (this being the reason for the sialogram being performed) and the sialectasis may thus be a result of the parotitis and not the cause of it. One has encountered sialectasis in a child after only one attack of parotitis (Fig. 18) and this has not been seen after a single attack of parotitis in adults, which does suggest that there is some difference between the two conditions.

It has been found that the sialectatic picture, seen in recurrent parotitis, is not due to dilatation of alveoli but to extravasation of radio-opaque material into the gland parenchyma through weak spots in the intraglandular ductules.[38] That this may also be the case in so-called congenital sialectasis is suggested by the finding in a boy of 9 with sialectasis after 5 attacks of parotitis (Fig. 19) that the radio-opaque material was still partially present 48 hours after the injection—an unlikely (though admittedly not impossible) event if the injected material was lying in dilated alveoli.

It is thus difficult at present to come to a definite conclusion about congenital sialectasis: histological proof of sialectasis occurring before any attacks of parotitis will be necessary to establish this condition as a definite entity. In the meantime it seems more likely that it is a condition of weakness of the finer ductules (allowing the extravasation of injected material to produce 'sialectasis') which may be acquired after parotitis or which is, more probably, due to some underlying congenital defect, as yet undetermined.

Branchial Cysts

The first branchial cleft is closely related to the developing parotid gland and the obliterated cleft takes a course from the external auditory canal, through the parotid gland, to just below the mandible. Sometimes the whole cleft may persist as a fistula traversing the gland but more commonly only a portion of the cleft remains patent to form a branchial cyst in the parotid gland. This latter condition has been encountered 3 times, (1) in a White female of 24, (2) in a Coloured male of 62 and (3) in a White female aged 13. They all presented with a symptomless cystic swelling in the parotid gland—of 2, 5 and 1 years' duration respectively. Theoretically one would expect these vestigial remnants to lie between the muscles of mastication and the trigeminal nerve (of the first arch) and the muscles of expression and the facial nerve (of the second arch) and thus deep to the facial nerve and its branches, but it seems to be superficial to the facial nerve in most cases[53] and has even been found to run between the branches of the nerve.[47] Sialography (and subsequent operation) showed 2 of my own 3 cases to be superficially placed (Fig. 20) but 1 was deep to the nerve (Fig. 21). The nerve must thus be carefully avoided during operations on this condition, and to this end sialography is a most useful investigation.

The cysts, as can be expected, contain fluid rich in cholesterol crystals and they are lined with squamous epithelium with lymphoid follicles in the wall (Fig. 24).

Vascular Malformations

The parotid salivary gland is extremely vascular and it is thus not surprising that vascular malformations constitute 0·5% of all swellings of the gland.[44] Such a lesion has been encountered only once by the author, in a White male aged 60 who had a mass in the deep part of the gland displacing the ducts laterally on sialography (Fig. 22). It was excised without damage to the overlying facial nerve and histologically it was an hamartomatous mass of maldeveloped blood vessels (Fig. 23).

SOME IMPORTANT STRUCTURAL FEATURES

Surgical Capsule

The parotid gland is enclosed in a tough fascial compartment which may be called a 'surgical capsule'. It makes palpation of the gland very difficult, and by virtue of its rigidity it disguises fluctuation in the gland; incision of an abscess in the gland should thus not be delayed until fluctuation can be elicited, because the tension inside this capsule is

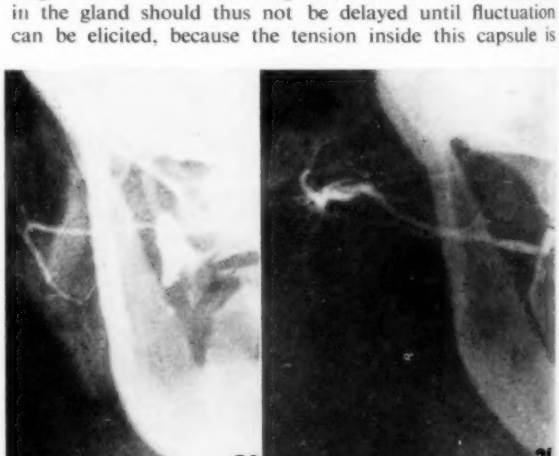

Fig. 20. Sialogram showing branchial cyst within parotid superfacial to facial nerve.
Fig. 21. Sialogram showing branchial cyst within parotid deep to facial nerve.

Fig. 22. Sialogram showing mass in the deep part of the gland displacing ducts laterally.
Fig. 23. Hamartoma of maldeveloped blood vessels (the mass shown in Fig. 22.)

responsible for extreme pain, destruction of the gland substance, and occasionally even damage to the facial nerve.

Parotid Lymph Glands

Inside this fascial capsule lie not only the parotid salivary gland but also lymphoid aggregations, most of which are histologically identical to ordinary lymph nodes.[52] Some authors have expressed their doubt whether those structures should be called lymph nodes,[32] but most anatomists accept them as such.[10, 17, 30] Fig. 25 illustrates a low-power view of such a lymphoid aggregation in the parotid salivary gland. These lymph glands are usually multiple and may be found superficial or deep to the salivary gland or actually within its substance.[15] They drain the greater part of the head and, as can be expected, they are liable to develop any of the diseases seen in other lymph glands, in which case a disorder of the parotid salivary gland will be simulated.[15] This has been encountered in 17 patients with the following conditions:

acute pyogenic adenitis—2 cases
chronic pyogenic adenitis—1 case
tuberculous adenitis—3 cases
lymphomata—6 cases
secondary carcinomata—5 cases

On occasions parotid salivary-gland inclusions are found in these parotid lymph glands,[35, 36] as can be seen on Fig. 26, and these inclusions are considered to be the origin of adenolymphomata, 2 of which I have myself seen.[15]

Parotid Salivary Gland

The parotid salivary gland is predominantly a serous gland. The alveolar cells synthesize secretion in their cytoplasm from raw material brought to them by the blood and tissue fluid. This secretion is dispersed in the cytoplasm in the form of tiny globules, which coagulate on fixation to form granules seen on microscopy.[18] After a period of rest the alveolar cells fill up with these granules, compressing the nucleus into an oval shape and pushing it into the basal part of the cell, but after secretion has taken place the cytoplasm is devoid of granules and the nucleus is larger, more spherical and in a more distal position in the cell.[50] This change in appearance of the cells with activity must be kept in mind to avoid its being misinterpreted as a pathological condition[14].

The parotid gland also contains some mucus-secreting cells.[49, 50] Some of them are present in the walls of the main ducts,[49] as can be seen in Fig. 27 stained with a P.A. Schiff stain to demonstrate mucus-secreting cells, and others appear as mucus-secreting acini (Fig. 28—also P. A. Schiff stain) or as semilunar crescents on serous acini.

The serous cells are stimulated to secrete by the parasympathetic nervous system *via* the auriculo-temporal nerve.[5] The mucous cells of the submandibular and sublingual salivary glands are stimulated to secrete mucus by the sympathetic nervous system.[5] No apparent secretion follows stimulation of the sympathetic supply to the parotid salivary gland,[5] but this may be only because there are so few mucus-secreting cells in the parotid that their secretions may not be readily noticeable, and it seems reasonable to assume that sympathetic stimulation does, in fact, produce mucus secretion by these mucous cells in the parotid salivary gland.

Many patients with recurrent parotitis have a markedly viscous saliva with a high mucus content. In some this viscosity is so high that every attempt at collecting the saliva by means of canulae of maximal size inserted into the duct failed because the saliva would not flow through

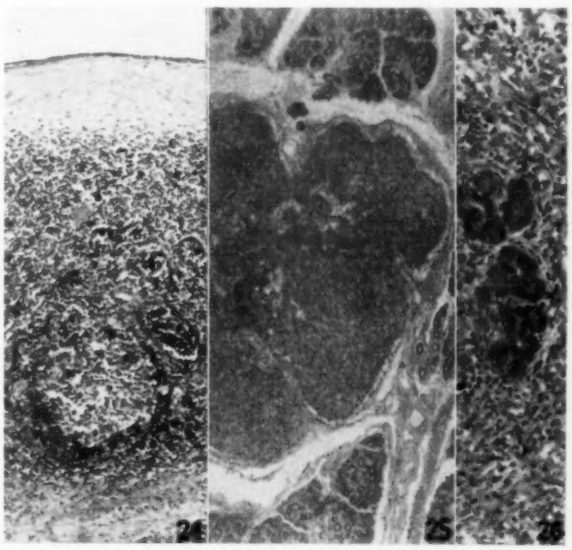

Fig. 24. Lining wall of branchial cyst showing lymphoid follicles and squamous epithelium.
Fig. 25. Lymphoid tissue in parotid gland.
Fig. 26. Salivary-gland inclusion in a parotid lymph gland.

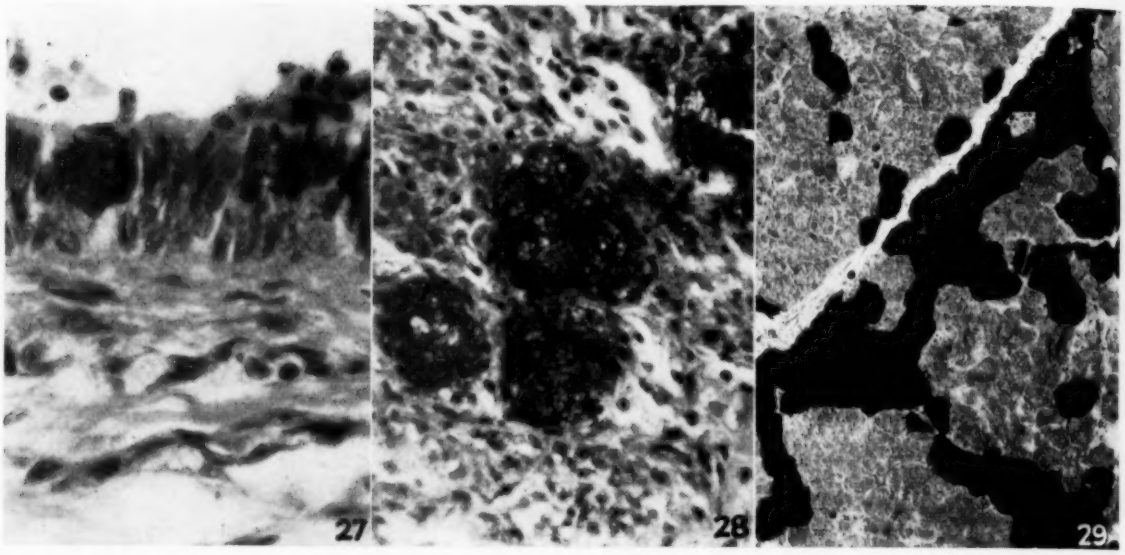

Fig. 27. Mucus-secreting cells in walls of main parotid duct. P. A. Schiff stain.
Fig. 28. Mucus-secreting acini within parotid gland. P. A. Schiff stain.
Fig. 29. Fat cells between acini of parotid gland. Sudan III.

the canulae. On 2 occasions the saliva was obtained in this manner after very great difficulty but was then found to be so viscous that it would not flow through a viscosimeter to measure its viscosity.

This increase of mucus in the saliva may be due to an increased number of mucous cells (this being congenital or acquired) or increased stimulation of a normal number of mucous cells (presumably *via* the sympathetic nervous system), and such cases must be interpreted in this way to obtain a correct diagnosis and to ensure the correct form of treatment.

The normally low mucin content of parotid saliva is responsible for the low incidence of salivary calculi in that gland as compared with the submandibular salivary gland (with its high mucin content) because a high salivary pH and a saliva rich in mucus are conducive to the deposition of the carbonates and phosphates of calcium in the saliva in the form of salivary calculi and tartar deposition on the teeth.[5] I have seen only 2 cases of parotid calculi, whereas submandibular calculi are a common occurrence.

Fat cells are found in the parotid gland in between the acini, as can be seen in Fig. 29 stained with Sudan III to show up the fat cells. An abnormal accumulation of these cells in obese people can produce diffuse enlargement of the parotid glands and on rare occasions a more localized mass, or even a lipoma, may be found.[6, 44]

Intralobular Ducts

The intralobular ducts of the parotid gland consist mostly of a single layer of cells, but between these and the basement membrane are scattered a few specialized cells—the myo-epithelial cells.[26] These cells are elongated, with their long axis parallel to the basement membrane, and they have an oval nucleus (Fig. 30). They can be shown up better on special staining, e.g. Masson's and von Gieson's stains, but this effect cannot be reproduced on black and white photographs. These cells are of some importance because under certain conditions they may proliferate in association with the epithelial cells to obstruct or even obliterate these fine ductules.

The terminal branches of the intraglandular ducts nearest the alveoli are sometimes called the intercalary ducts, and in the parotid gland these are longer and narrower than those of the submandibular salivary gland.[42] This narrow intercalary duct is clearly seen on a high-power view of the gland (Fig. 31), and will obviously interfere to some extent with the free flow of saliva; this has been blamed for the increased susceptibility of the parotid salivary gland to infection.[24, 46] This structural difference between the two salivary glands is also responsible for a significant difference seen on sialography. If a large volume of radio-opaque material is injected into the submandibular duct it readily enters the alveoli, but in the parotid glands the narrow intercalary ducts produce some slight obstruction to the free flow of the injected material. This results in an increased pressure at the level of the smaller ductules, which may rupture under the strain,[42] particularly if they are diseased, to produce the picture of sialectasis, which is thus commoner in the parotid than in the submandibular salivary gland.

The Interlobular Ducts

These ducts are lined by columnar epithelium with rounded or elliptical intermediate cells and one or more layers of rounded basal cells.[49] These basal cells are pluripotent and can differentiate into columnar cells or into mucous cells, and the columnar cells themselves can also assume mucus-secreting properties to form fully developed goblet cells.[49] It is thus not unexpected that after long-continued infection there may be such a marked proliferation of mucus-secreting goblet cells (Fig. 32) that the saliva contains a

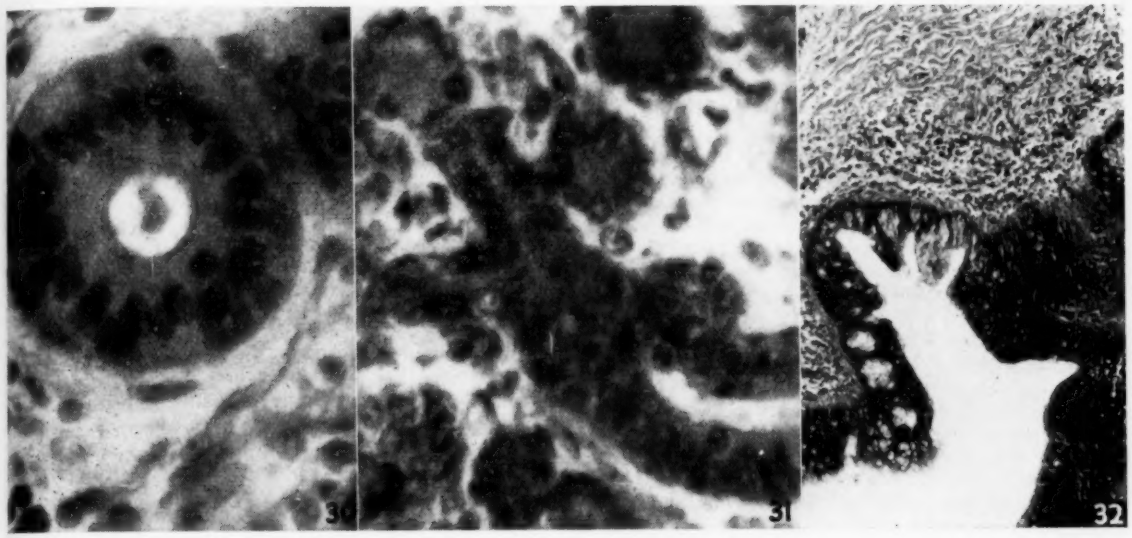

Fig. 30. Myo-epithelial cells in intralobular duct.
Fig. 31. Intercalary duct in parotid gland.
Fig. 32. Parotid gland showing mucus-secreting goblet cells in a case of long-continued infection.

significant proportion of mucus and is consequently much more viscous than normal. This is an important effect of recurrent inflammation since the viscous saliva will markedly interfere with the free flow of saliva and consequently with the discharge of pus from the gland.

The Main Extraglandular Duct

This duct is longer and narrower than the main submandibular duct, and the stasis resulting from this is another factor in the increased susceptibility of the parotid gland to infection.[2, 45] It has smooth muscle in its wall and it has been held that there is a concentration of this muscle around the oral termination of the duct where it appears to form a sphincter.[37] This one has not been able to confirm but there is no doubt that the parotid duct orifice is the narrowest portion of the duct[17] and that consequently the duct tends to act as a reservoir for saliva.[37]

The saliva so retained in the duct is squeezed out into the mouth during talking and chewing because within ½ inch of the orifice the duct bends sharply to the lateral side and pierces the muscles of the cheek. This again emphasizes the importance of talking and chewing in preventing stasis of saliva. The tortuosity of the duct should also be remembered when performing a sialogram or probing the duct because, if the cannula or probe is inserted straight, its end will impinge on the far wall of the duct at the bend to simulate a stricture, for the dye will flow back into the mouth and the probe will not pass beyond this spot.

The Parotid Duct Orifice

The duct orifice is situated in a cul-de-sac under the cheek superior to the level of the cusps of the second upper molar tooth. In this situation it is particularly liable to injury during chewing, especially in those people who have the duct opening on the tip of a little papilla. More frequently, however, this type of injury occurs when the upper molars are lower than normal thus bringing the chewing surface level with the duct orifice. This occurs in children during the eruption of the second molars,[45] in adults with ill-fitting dentures or in older people who have worn dentures for many years with the resultant atrophy of the gums lowering the cusp level of the molars. Any such injury will produce oedema (with a temporary obstruction to the flow of saliva) and later fibrosis (with a permanent orifice stenosis).

Being situated in a cul-de-sac far away from the cleansing action of the tongue during talking and swallowing, the area around the parotid duct orifice will become very dirty, particularly in people who are seriously ill and who thus do not consciously clean the mouth regularly. This cleansing effect of swallowing is most marked under the tongue and consequently the area around the submandibular duct orifices is constantly kept clean. This difference has been demonstrated by insufflating charcoal powder into the mouths of normal people: after an average of 4 minutes of normal activity the area under the tongue was cleansed of the charcoal but the area around the parotid duct orifice was still heavily contaminated with charcoal after an average of 20 minutes. It is further confirmed by the report that in the post-operative period swabs taken from the region of the parotid duct orifice grew coagulase-positive *Micrococcus pyogenes aureus* far more frequently than those taken from under the tongue.[9, 40] One has been unable to confirm this latter finding, swabs taken from these two sites having always shown similar results on culture, but nevertheless this may be yet another factor responsible for the high incidence of post-operative parotitis as opposed to infection of the submandibular salivary gland.

IMPORTANT ASPECTS OF PAROTID GLAND FUNCTION

Under normal conditions bacteria enter the parotid duct from the mouth and are carried towards the gland,[7] with the result that bacteria similar to those in the mouth[8] can regularly be found in the duct.[11] This is naturally more severe if the mouth is dirty, but one has often seen a dirty mouth without the development of parotitis and it is obvious that a most efficient protective mechanism exists to prevent the onset of parotitis. This is confirmed by the reports that contaminating the parotid duct orifice with pathogenic organisms does not produce parotitis, [7, 43] and even injecting bacteria into the parotid duct produces parotitis in only 50% of animals.[4]

It is often held that this defence against parotitis depends on the bactericidal effect of the saliva itself,[40] which is thought to be due to the mucus in the saliva[8]—this would also explain the low incidence of spontaneous infection in the submandibular salivary gland, which has a higher mucin content in its saliva than the parotid gland.[8, 46, 51] With the kind assistance of Drs. D. McKenzie and A. Naude, of the Department of Bacteriology, we have attempted to demonstrate this bactericidal effect of saliva by placing parotid and submandibular saliva on plates seeded with *M. pyogenes aureus*, but no inhibition of growth of this organism could be demonstrated; we doubt whether the bactericidal effect of saliva is of any great significance.

The main defence of the gland against ascending infection is the flushing effect of normal salivary flow, and anything which interferes with this will predispose to the occurrence of parotitis. Salivary flow depends on adequate hydration (the production of saliva is decreased during dehydration), regular stimulation of salivary secretion (as occurs with eating, chewing[41] and talking), and a free passage of the saliva along the parotid duct into the mouth, to prevent stasis of the saliva in the gland or duct. Thus, while it is clearly a wise precaution to maintain a clean mouth post-operatively or during a serious illness, in an attempt to prevent parotitis, it is of far greater importance to maintain free salivary flow by ensuring good hydration, regular stimulation of the gland (by chewing gum and sucking acid-drops) and an unobstructed duct system.

SUMMARY

The manifestations of disease in the parotid salivary glands are largely controlled by developmental, structural and functional factors and consequently an understanding of these is essential for a correct appreciation of pathology and treatment.

The development of the parotid salivary glands is described, and various congenital abnormalities are discussed, such as accessory parotid glands, congenital stenosis, congenital displacement, congenital sialectasis, branchial cysts and vascular malformations.

Various structural peculiarities are mentioned, with an emphasis on their practical importance; this includes the parotid capsule, intraglandular lymph glands, parenchymal structure, intra- and extraglandular duct structure and the parotid duct orifice.

Lastly, the mechanism of protection against ascending infection is discussed, with particular stress on the prevention of parotitis.

The author wishes to thank the many colleagues who have referred these patients for treatment, the Superintendent of Groote Schuur Hospital for permission to report the cases, and Prof. J. H. Louw for the many facilities he has provided during these investigations.

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FAMILIAL UNILATERAL GYNAECOMASTIA IN AN ADOLESCENT

THEODORE JAMES

Pinelands, Cape

The following case of unilateral familial gynaecomastia is reported:

The patient was 17 years old when he asked for medical advice about the excessive development of the soft tissues of his right breast. It was after he had turned 15 that he first detected the beginning of this abnormality on the right side only; on the left side no similar manifestation had ever suggested itself. From the onset it had progressed continuously until the time of the interview, when he wished for something to be done about it.

The physical examination was essentially normal for a youth of his age excepting for the local condition. A remarkable degree of gynaecomastia was present on the right side. It was comparable with the breasts of a young primipara of asthenic type. Subcutaneous mammary tissue was readily palpable, the nipple was larger than the left nipple and the areola was also relatively bigger and a shade darker. The youths' medical history was excellent and he had no treatment which could have had any relationship to his condition. Despite the very normal-looking external genitalia, he was closely questioned to verify the presence of average potency and spermatogenesis for his age; the answers were satisfactory.

Fig. 1

Because the gynaecomastia had persisted over-long and still showed no sign of regression, the associated psychical distress was regarded as justification for removal of the unwanted tissue. When this procedure was advised the patient volunteered the information that an elder brother had been operated upon some years before for the same complaint. The brother, who was a few years older than the patient, had shown signs of gynaecomastia at the age of 8 years. His condition also had been progressive until surgical removal. There were no other siblings in the family and the patient knew of no close male relations with the condition of gynaecomastia.

DISCUSSION

This short case-history has stimulated enquiry into the possible causes of abnormal development of breast tissue in the male. Gynaecomastia has been defined as an excessive size of the male mammary glands, and by its derivation simulation of the female mamma is implied. The immediate relation of secondary sex characteristics to the sex hormones suggests a female hormonal influence over-active in the male producing gynaecomastia. This postulate has given rise to much clinical observation and experimental investigation to confirm the association of anatomy and hormone and from this research one learns to distinguish as physiological the swelling of the breast tissues in the newborn and at puberty in both sexes from the mastopathy of later life.

With the onset of puberty in the boy it is not uncommon for the normal pubertal development to be accompanied by a slight swelling of the breast tissue on both sides and sometimes, as in this case, on one side only. This is comparable with the early development in the female. The frequency of this growth-manifestation has been put at 20% in healthy adolescent boys, including slight as well as marked increase of the breast tissue. The duration of the adolescent gynaecomastia averages 17 months and it subsides completely about the 17th year. Often, however, subareolar nodules persist until well on into adult life.

Perhaps it is true to say that even today the exact biological interplay of hormones that produces first the mammary maturation and then the secretion of milk is not completely understood. This fact adds to the difficulty of finding an adequate explanation for adolescent gynaecomastia and still more so when the gynaecomastia is unilateral. It is believed now that two hormones, follicular hormone and progesterone, are essential for the full growth of the breast. It is assumed that there is a probable relation between the gynaecomastia of puberty and a disproportionate secretion of androgen and oestrogen in the male so that the ratio of the secretion

of the two substances in gynaecomastia of adolescence approaches that in the normal adolescent female.

It is not relevant to enter into any details concerning mammary changes associated with disease of the male genital organs except to mention that when breast changes do occur with such diseases an increase of oestrogen above the normal level in the male is often demonstrable.

Forssell[1] concluded after observing very many newborn babies that a mammary secretion occurred in 99% of both male and female babies born at term. It is established that normal newborn infants have an excess of oestrogens in their blood, and the milk secreted in the breast of the new-born infant has been chemically shown to have many of the properties of colostrum.[2] This appears to have a direct bearing upon another fact, namely, that prolactin appears before and during the time of the infant's lactation and that the quantity varies with the amount of secretion. Whereas it is accepted by endocrinologists that the luteal hormones formed in corpus luteum and placenta are necessary for the proliferation of the secretory tissue of the breast, nevertheless lactation has been induced in babies past the neonatal age by the exhibition of, first, oestrogen and then prolactin.[3]

CONCLUSION

There is very little about gynaecomastia in the text-books; where it is mentioned in any detail it forms part of the syndrome of a pathological picture like that, say of an adrenal-gland tumour. The information presented in this paper provokes speculative thinking on what, in persistent cases, appears to be an aberrant manifestation. In those instances of pubertal gynaecomastia which resolve spontaneously before the end of adolescence, it is a reasonable assumption that during these years of intensive hormonal activity a relative and disproportionate increase of oestrogen substances from the adrenal gland causes an enlargement of the breasts which regresses when the normal adjustment of the hormonal secretions becomes established. That other signs of an excess of oestrogen production are not obvious clinically may be explained by the supposition that androgens predominate and maintain the distinct sex difference and that the breast tissues are especially sensitive to even slight increases of oestrogen above the normal.

The unilateral gynaecomastia is not so plausibly explained. However, the fact that not infrequently there is a marked difference in the size of the two breasts in pubertal gynaecomastia suggests that variations exist in the amount of responsive mammary tissue. When the condition is unilateral, it may be surmised that there is no mammary tissue on the normal side. Against this surmise is the fact that in none of the series of clinical observations on the so-called (and miscalled) 'neonatal mastitis' or enlargement with lactation of the breasts in the newborn has any mention been made of unilateral swelling in male infants. The possibility remains that observations of unilateral neonatal swelling may have been made without being recorded. Certain of these series of neonatal observations have been quite considerable[1, 4], which suggests that it would be of value to observe a large series of male neonates for unilateral swelling and lactation of the breast.

In the relatively few cases where the breast enlargement does not regress after adolescence, if the gynaecomastia is due to a relative excess of oestrogen substance, is the persistence of this relative excess the cause of the persistent gynaecomastia? Or, if the postulated excess of oestrogen substance disappears and normal hormone levels for the male become established, does an unresolved gynaecomastia imply that irreversible breast changes have come about?

The familial incidence of this case of gynaecomastia deserves to be recorded, for a search through the literature has not uncovered another instance. Although the unilateral state has happened in two full brothers, this frequency in one family does not exceed that of pure chance, but a genetic influence is suggested, by the fact that the identical abnormality has occurred in the only two brothers, it being unilateral in both, and persistent in both to the point of requiring surgical removal.

SUMMARY

A short case-history of unilateral and familial gynaecomastia in an adolescent is presented. The probable aetiological factors behind this condition in youthful males are considered. From theory and clinical evidence the author submits his speculative conclusions for further consideration.

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ALKAPTONURIA IN TWO SISTERS

W. M. POLITZER, M.D.

South African Institute for Medical Research, Johannesburg

and

CORNELIA J. REITZ, M.B., B.CH.

No. 1. *Military Hospital, Voortrekkerhoogte, Pretoria*

Alkaptonuria is a metabolic disorder characterized by alkaptone (homogentisic acid) in the urine. When the urine is passed and is acid it is of normal colour but when passed alkaline it is brown; when left standing or when alkali is added, it becomes black. This condition can be a Mendelian recessive[1] or a Mendelian dominant.[2, 3]

It is a very rare disorder and fewer than 200 cases have been reported in the world literature.[4] Up to the age of

adolescence there are usually no signs or symptoms except the dark urine. However, in the third decade of life, bluish-black discolouration and rigidity of the auricular and nasal cartilages occur as the first sign of ochronosis, and subsequently osteoarthritis and arthropathies develop.

CASE REPORT

Mrs. S., a 19-year-old housewife 26 weeks pregnant, was attending the antenatal clinic of the Voortrekkerhoogte Hospital. On routine examination of the urine for sugar with Benedict's reagent, a greenish-brown discolouration was obtained. On enquiry, the patient recollected that her mother had mentioned that as a baby the patient's napkins were frequently stained dark-brown. The patient had also noticed that her urine became dark-brown on standing. Further tests were carried out in order to identify the reducing substance in the urine and this was found to be homogentisic acid.

She was a healthy-looking well-nourished young woman. She was fair-skinned and freckled and had red hair and brown eyes. On examination a faint blue discolouration was seen over both elbows, and no other pigmentation of the skin or of the mucous membranes was found. The ear cartilages were somewhat rigid. The spine and all joints were fully mobile. No abnormalities were detected in the cardio-vascular, respiratory and central nervous systems. The blood pressure was 120/70 mm. mercury. Neither liver nor spleen was palpable. The uterus was enlarged to a ±26 weeks pregnancy.

At full term the patient was delivered of a normal living male infant and her puerperium was uneventful. At this time X-rays of the chest, spine and of all joints, and electrocardiograms, were taken and found to be completely normal. Ophthalmoscopic and laryngoscopic examinations were performed and no abnormal pigments were observed.

Laboratory Investigations

Repeated quantitative homogentisic acid determinations on 24-hour specimens of urine by the method of Neuberger[5] gave the following results: 3·5, 2·8, 3·3 g. The blood Wassermann test was negative. The haemoglobin, red blood count, blood urea, urea clearance, glucose-tolerance test, serum uric acid, cholesterol, potassium, sodium and phosphorus were all normal. The only abnormal laboratory finding was evidence of impaired liver function during the pregnancy but normal results were obtained after parturition.

In view of the fact that this disorder occurs in families, urines from all available relatives were obtained; they were found to be negative with one exception, viz. the patient's 11-year-old sister from whom a 24-hour specimen of urine contained 3·2 g. of homogentisic acid.

Family History

A distinctive feature of these 2 cases is the complete absence of a history of alkaptonuria in their forbears. Neither paternal nor maternal parents or grandparents showed any signs of alkaptonuria, ochronosis or of arthritis, nor were the patients' 16- and 18-year-old brothers affected. Enquiries were made and all available records show that there had been no intermarriage. The urine of the patient's newborn child was repeatedly examined for homogentisic acid but the results were negative.

DISCUSSION

Homogentisic acid is not normally excreted in urine. In normal metabolism phenylalanine and tyrosine are oxidized by way of homogentisic acid to aceto-acetic acid and finally to carbon dioxide and water.

In the patient with alkaptonuria the enzyme necessary for oxidizing homogentisic acid (homogentisic-acidase) appears to be lacking, and homogentisic acid is excreted in the urine.

These two cases appear to be the first reported in South Africa. As a rule the condition is hereditary but in this family it has not been possible to demonstrate any genetic pattern. Similar cases without a family history have been reported.[6, 7] It would seem therefore that heredity is not always a predisposing factor.

We wish to express our thanks to Dr. I. Bersohn for the liver function tests and to Dr. J. L. van Staa for the quantitative homogentisic-acid determination.

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TOBACCO SMOKING AND CANCER OF THE LUNG

STATEMENT BY THE BRITISH MEDICAL RESEARCH COUNCIL *

In its annual report for 1948-50† the Council drew attention to the very great increase that had taken place in the death rate from lung cancer over the previous 25 years. Since that time the death rate has continued to rise, and in 1955 it reached more than double that recorded only 10 years earlier (388 deaths per million of the population in 1955 compared with 188 in 1945). Among males the disease is now responsible for approximately 1 in 18 of all deaths. Although the death rate for females is still comparatively low, it also has shown a considerable increase in recent years, and the disease is now responsible for 1 in 103 of all female deaths.

Three comments may be made on these figures. In the first place, the trend over the last few years indicates that the incidence has not yet reached its peak. Secondly, the figures are not to be explained as a mere reflection of the introduction and increasing use of improved methods of diagnosis but must be accepted as representing, in the main, a real rise in the incidence of the disease to an extent which has occurred with no other form of cancer. Thirdly, only a small part of the rise can be attributed to the larger numbers of older persons now living in the population; in the last ten years the lung cancer death rates among both men and women have risen at all ages from early middle life onwards.

POSSIBLE CAUSES OF THE INCREASE

The extent and rapidity of the increase in lung cancer point clearly to some potent environmental influence which has become prevalent in the past half-century and to which different countries, and presumably also men as compared with women, have been unequally exposed. The pattern of incidence of the disease rules out any possibility that the increase can be due, in a substantial degree, to special conditions, such as occupational hazards, affecting only limited groups. It is necessary to seek some factor or factors distributed generally throughout the population, and in considering the possibilities it must be borne in mind that a very long period, 20 years or more, may elapse between exposure to a carcinogenic agent and the production of a tumour. From the nature of the disease attention has focused on two main environmental factors: (1) the smoking of tobacco, and (2) atmos-

* Reprinted from Brit. Med. J., 29 June 1957 (**1**, 1523).
† *Cmd.* 8387 (1951): London: H.M. Stationery Office.

pheric pollution—whether from homes, factories, or the internal combustion engine.

SMOKING AS A CAUSE OF LUNG CANCER

(a) *Epidemiological Surveys*

The evidence that heavy and prolonged smoking of tobacco, particularly in the form of cigarettes, is associated with an increased risk of lung cancer is not based on the observation that the substantial increase in the national mortality followed an increase in the national consumption of cigarettes. It is derived from two types of special inquiry. In the first, patients with lung cancer have been interviewed and their *previous* histories in relation to smoking and other factors that might be relevant have been compared with those similarly obtained from patients without lung cancer. The results of nineteen such inquiries (in this country, the U.S.A., Finland, Germany, Holland, Norway and Switzerland) have been published. They agree in showing more smokers and fewer non-smokers among the patients with lung cancer, and a steadily rising mortality as the amount of smoking increases. In the second type of inquiry, information has been obtained about the smoking habits of each member of a defined group in the population and the causes of the deaths occurring *subsequently* in the group have been ascertained. There have been two such investigations, one in the U.S.A. covering 190,000 men aged 50-69, and the other in Great Britain covering over 40,000 men and women whose names appeared on the *Medical Register* of 1951. In both the results have been essentially the same. The investigation in Great Britain, which has now been in progress for more than five years, has shown with regard to lung cancer in men: (1) a higher mortality in smokers than in non-smokers; (2) a higher mortality in heavy smokers than in light smokers; (3) a higher mortality in cigarette smokers than in pipe smokers; and (4) a higher mortality in those who continued to smoke than in those who gave it up.

It follows that the highest mortalities were found among men who were continuing to smoke cigarettes, heavy smokers in this group having a death rate nearly 40 times the rate among non-smokers. Although no precise calculation can be made of the proportion of lifelong heavy cigarette smokers who will die of lung cancer, the evidence suggests that, at current death rates, it is likely to be of the order of 1 in 8, whereas the corresponding figure for non-smokers would be of the order of 1 in 300. The observation on the effect of giving up smoking is particularly important, since it indicates that men who cease to smoke, even in their early forties, may reduce their likelihood of developing the disease by at least one-half.

It should be noted that the excess of deaths from lung cancer among smokers was not compensated for by any corresponding reduction in the number of deaths from cancer of other sites in the body; in other words, there was a total incidence of cancer in the smoking groups in excess of the incidence that would have prevailed in the absence of smoking.

It will be apparent from what has been said that the evidence from the many inquiries in the last eight years, both in this country and abroad, has been uniformly in one direction and is now very considerable. It has been further strengthened recently by the observation from several sources that the extent of the relationship with smoking differs for different types of lung tumour which can be distinguished only by microscopical examination.

(b) *Laboratory Evidence*

From the physical and chemical point of view there is nothing inherently improbable in a connection between smoking and lung cancer. Tobacco smoke consists largely of microscopic oily droplets held in suspension in air, and these droplets are of a suitable size to be taken into the lungs and retained there. Over a hundred constituents have so far been identified, and, among these, five substances have already been found which are known to be capable, in certain circumstances, of causing cancer in animals. Some workers have succeeded in producing tumours in animals by painting concentrated extracts of tobacco tar on the skin. Known carcinogens are present in tobacco smoke in very small amounts, however, and there is no certainty that such low concentrations could be harmful to human beings. Nevertheless, the finding of carcinogenic agents in tobacco smoke is an important step forward, in that it provides a rational basis for the hypothesis of causation.

ATMOSPHERIC POLLUTION AS A CAUSE OF LUNG CANCER

It has been known for some years that mortality from lung cancer is greater in urban areas than in the countryside. This fact, together with the identification of carcinogenic substances in coal smoke and in motor vehicle exhausts, has led to the supposition that exposure to atmospheric pollution may be concerned with the increase in lung cancer. The role of atmospheric pollution is particularly difficult to investigate however, and the evidence is neither so consistent nor so extensive as that relating to tobacco smoking. On the one hand, no excess mortality from lung cancer has been observed in persons who would be especially exposed by the nature of their work to atmospheric pollution—for example transport workers, garage hands, and policemen. On the other hand, the results of a number of investigations have suggested that a relationship does exist between atmospheric pollution and lung cancer. Perhaps the best evidence for this relationship comes from studies of the small numbers of deaths from the disease among *non-smokers* in different types of residential district; in these studies higher death rates have been observed among non-smokers in large towns than among those in rural areas. On balance, it seems likely that atmospheric pollution plays some part in causing the disease, but a relatively minor one in comparison with cigarette smoking.

ASSESSMENT OF THE EVIDENCE RELATING TO SMOKING AND LUNG CANCER

Knowledge of the causation of lung cancer is still incomplete. Many factors other than tobacco smoking are undoubtedly capable of producing the disease; for example, at least five industrial causes have been recognized. Nevertheless, the evidence for an association between lung cancer and tobacco smoking has been steadily mounting throughout the past eight years, and it is significant that, during the whole of this period, the most critical examination has failed to invalidate the main conclusions drawn from it. It has indeed been suggested that the fundamental cause may be some common factor underlying both the tendency to tobacco smoking and to the development of lung cancer some 25 to 50 years later, but no evidence has been produced in support of this hypothesis.

In scientific work, as in the practical affairs of everyday life, conclusions have often to be founded on the most reasonable and probable explanation of the observed facts, and so far no adequate explanation for the large increase in the incidence of lung cancer has been advanced save that cigarette smoking is indeed the principal factor in the causation of the disease. The epidemiological evidence is now extensive and very detailed, and it follows a classical pattern upon which many advances in preventive medicine have been made in the past. It is clearly impossible to add to the evidence by means of an experiment in man. The Council are, however, supporting a substantial amount of laboratory research which may throw more light on the mechanism by which tobacco smoke and other suspected causative factors exert their effect, and which may thus eventually add to the degree of proof already attained as a result of studies of human populations. It must be emphasized, however, that negative results from work with animals cannot invalidate conclusions drawn from observations on man.

CONCLUSIONS

1. A very great increase has occurred during the past 25 years in the death rate from lung cancer in Great Britain and other countries.

2. A relatively small number of the total cases can be attributed to specific industrial hazards.

3. A proportion of cases, the exact extent of which cannot yet be defined, may be due to atmospheric pollution.

4. Evidence from many investigations in different countries indicates that a major part of the increase is associated with tobacco smoking, particularly in the form of cigarettes. In the opinion of the Council, the most reasonable interpretation of this evidence is that the relationship is one of direct cause and effect.

5. The identification of several carcinogenic substances in tobacco smoke provides a rational basis for such a causal relationship.

FEDERAL COUNCIL ELECTIONS

The results of the recent 5-yearly Branch elections for Federal Council which have been received at the time of going to press, show that the following candidates have been elected:

Cape Western
J. C. Coetzee.
J. A. Currie
J. H. Dommisse.
M. Helman.
A. Landau.
T. B. McMurray.
J. A. S. Marr.
P. F. Oates.
A. G. Paterson.
J. H. L. Shapiro.
A. W. S. Sichel.
L. Slabbert.
A. A. Zabow.

East Rand
D. E. McKenzie.
M. Segal.
E. W. Turton.

Natal Inland
B. A. Armitage.
T. H. Whitsitt.

Northern Transvaal
J. H. Casewell.
C. M. Grundlingh.
W. H. Lawrance.
J. H. Struthers.
W. Waks.
F. Ziady.

Southern Transvaal
C. Adler.
A. L. Agranat.
N. E. C. de la Hunt.
W. Girdwood.
J. Gluckman.
S. C. Heymann.
H. Penn.
T. Radloff.
L. S. Robertson.
T. Schneider.
M. Shapiro.
L. O. Vercueil.
J. Wolfowitz.

South West Africa
W. H. G. Kuschke.

CAPE TOWN PROTEST MARCH

A Special General Meeting of the Cape Western Branch of the Medical Association of South Africa was convened in the Physiology Lecture Theatre, Medical School, Observatory, Cape, on Tuesday 23 July 1957.

This meeting was requisitioned as a matter of urgency by 10 members of the Branch in order to discuss a letter received by these members and others of the Association from the Superintendent of Groote Schuur Hospital, Cape Town. This letter requested them to reply to him by noon on 24 July, stating whether or not they had taken part in the Universities Protest Procession, on 7 June 1957.

The meeting was attended by about 180 members—chiefly from the Cape Town and Northern Areas Divisions.

After considerable discussion the following two motions were passed:

(*a*) That this meeting recommends that members should acknowledge receipt of the Superintendent's circular and state that the contents have been noted—*passed by a large majority.*

(*b*) That a deputation consisting of 3 senior members of the Association chosen by the meeting be asked to interview the Administrator in order to clear up this impasse in a friendly and dignified way—*passed nem con.*

The following 3 members of the deputation were then elected by ballot:

Mr. J. A. Currie, Mr. R. D. H. Baigrie and Prof. James T. Louw. With the consent of the meeting Dr. M. Jordaan was coopted to this deputation.

The interview with his Honour the Administrator (Mr. P. J. Olivier) took place on 25 July at his residence Leeuwenhof, Cape Town. He was attended by the Director of Hospitals (Dr. D. A. van Binnendyk) and Mr. J. N. Malan, M.E.C., and all four appointed members of the deputation were present.

Discussions took place in a cordial atmosphere. The Administrator intimated his willingness to receive a deputation from the Medical Staff of Groote Schuur Hospital.

THE MENTAL DISORDERS AMENDMENT ACT 1957

This Act[1] (No. 37 of 1957), which was gazetted under Government Notice No. 892 of 21 June 1957, effects further amendments to the Mental Disorders Act, No. 38 of 1916 (referred to as the 'principal Act') as follows:

Section 1 amends section 3 of the principal Act (as amended in 1944) by the deletion of the description of Class VI of mentally disordered or defective persons. [This description was enacted by the Mental Disorders Amendment Act, No. 7 of 1944 as follows: 'ClassVI.—A socially defective person, that is to say, a person who suffers from mental abnormality associated with anti-social conduct, and who by reason of such abnormality and conduct requires care, supervision and control for his own protection or in the public interest.']

Section 2 amends section 43 of the principal Act (which provides for the detention of persons declared by 'warrant' to be mentally disordered or defective, who have been admitted into the Union from a 'proclaimed' African territory with the previous consent of the Minister) by the following paragraph, the existing sub-section becoming paragraph (*a*):

'(*b*) The Minister shall not grant such consent in respect of any person who is not domiciled in the Union, unless the government of the proclaimed territory has undertaken to pay the maintenance and other expenses which may be incurred by the Government of the Union in connection with the detention of such person in an institution.'

Section 3 amends section 53 of the principal Act (as amended in 1944) so as to alter the titles of the Commissioner and the Deputy Commissioner of mentally disordered and defective persons to 'Commissioner (or Deputy Commissioner) for mental hygiene'.

Section 4 substitutes a new section for section 79 of the principal Act, which provided that the cost of maintaining (etc.) persons detained under the Act should be defrayed out of the consolidated revenue fund, and that the cost might be recovered from their estates or from persons liable by law to contribute to their maintenance. The new section is as follows:

'79. (1) Subject to the provisions of sub-section (2), the maintenance and other expenses necessarily incurred in connection with the detention by the Government in any institution under the provisions of this Act, of any person, other than a person in respect of whom the provisions of section 43* or 80† apply, shall be defrayed out of monies appropriated by Parliament for the purpose.

(2) The said maintenance and other expenses—

(*a*) in respect of any person detained under section 44‡ or 52§; or

(*b*) which in terms of any law shall be borne by the Administration of the territory of South West Africa,

* Concerning mental patients from a 'proclaimed' African territory.

† Concerning mental patients from on board ship or aircraft.

‡ 'Voluntary boarders'.

§ Certain other patients dealt with like voluntary boarders.

may be recovered from the estate of the person detained or from any person liable by law to contribute towards his maintenance.'

[The effect of this amendment is (subject to certain exceptions) to bring to an end the levying on relatives etc. of charges for the maintenance of persons detained in mental hospitals.[1]]

Section 5 amends section 80 of the principal Act:

(*a*) by the substitution for the word 'port', wherever it occurs, of the words 'seaport or airport';

(*b*) by the insertion after the word 'vessel' wherever it occurs, of the words 'or aircraft';

(*c*) by the substitution for the words 'or master', wherever they occur, of the words 'master or pilot'; and

(*d*) by the substitution in sub-section (2) for the words 'whence he was shipped' of the words 'where he boarded such vessel or aircraft'.

[The effect is to extend the procedure concerning mental patients entering the Union from ships to those doing so from aircraft.]

Section 6 amends section 86 of the principal Act (which enables the Governor General to make regulations) by the substitution for paragraph (*e*) of subsection (1), concerning terms for paying patients, of the following paragraph:

'(*e*) the payment of maintenance and other expenses necessarily or otherwise incurred in connection with the detention and maintenance of any person in any institution other than a licensed institution'.

Section 7. Definitions.

Section 8. Short title.

1. Report (1957): S. Afr. Med. J., **31**, 544.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

MEDICAL ASSOCIATION OF SOUTH AFRICA : ANNUAL GENERAL MEETING

Notice is hereby given that the Annual General Meeting of the Medical Association of South Africa will be held in the B.E.S.L. Hall, Old Fort Road, Durban, on Wednesday 11 September 1957, at 9.30 a.m.

Agenda

1. Minutes.
2. Annual Report and Balance Sheet.
3. Election of Auditors.
4. Induction of President.
5. Other Business.

At the conclusion of business, the meeting will be adjourned and reconvened on Monday 16 September 1957, at 8 p.m. in the City Hall, Durban, where, combined with the Opening Ceremony of Congress, the President will deliver his Presidential Address. Academic dress will be worn.

A. H. Tonkin
Secretary

Medical House
Cape Town
22 July 1957

MEDIESE VERENIGING VAN SUID-AFRIKA : ALGEMENE JAARVERGADERING

Kennis geskied hiermee dat die Algemene Jaarvergadering van die Mediese Vereniging van Suid-Afrika gehou sal word op Woensdag 11 September 1957, om 9.30 vm., in die B.E.S.L. Saal, Old Fort-weg, Durban.

Agenda

1. Notule.
2. Jaarverslae.
3. Verkiesing van Ouditeure.
4. Inleiding van President.
5. Ander Besigheid.

Na afhandeling van dié werksaamhede, word die vergadering verdaag en op Maandag 16 September 1957 om 8 nm. in die Stadsaal, Durban, hervat, wanneer die Openingsplegtigheid van die Kongres plaasvind en die President sy voorsittersrede sal lewer. Akademiese drag word gedra.

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
22 Julie 1957

FEDERAL COUNCIL

Notice is hereby given that a meeting of the Federal Council will be held in the B.E.S.L. Hall, Old Fort Road, Durban, on 11, 12 and 13 September 1957, commencing at 10 a.m.

Agenda

1. Notice convening the meeting.
2. Proxies.
3. Minutes of previous meeting (circulated).
4. Matters arising out of the minutes.
5. Financial statement by Honorary Treasurer.
6. Report of the Executive Committee.
7. Reports of other Committees.
8. Reports deferred from previous meeting.
9. Notices of motion transferred from previous meeting.
10. New notices of motion.
11. Other business.

A. H. Tonkin
Secretary

Medical House
Cape Town
22 July 1957

FEDERALE RAAD

Kennis geskied hiermee dat 'n vergadering van die Federale Raad gehou sal word in die B.E.S.L. Saal, Old Fort-weg, Durban op 11, 12 en 13 September 1957, aanvang 10 vm.

Agenda

1. Kennisgewing wat die vergadering belê.
2. Volmagte.
3. Notule van die vorige vergadering (reeds uitgestuur).
4. Sake wat uit die notule voortspruit.
5. Finansiële verslag van die Ere-Penningmeester.
6. Verslag van die Uitvoerende Komitee.
7. Verslae van ander Komitees.
8. Verslae van vorige vergadering oorgehou.
9. Voorstelle waarvan kennis op vorige vergadering gegee was.
10. Nuwe kennisgewings van voorstelle.
11. Ander sake.

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
22 Julie 1957

THE BENEVOLENT FUND : DIE LIEFDADIGHEIDSFONDS

The following contributions to the Benevolent Fund during April, May and June 1957, are gratefully acknowledged.

Votive Cards in Memory of:

Dr. R. D. Osler *by* Mr. F. K. Mitchell.
Dr. C. Comrie Sharp *by* Dr. J. S. du Toit.
Dr. H. M. Griffiths *by* Dr. and Mrs. A. W. Sichel.
Dr. Gordon J. Key *by* Drs. W. P. and T. P. S. Mulligan.
Mrs. A. Jocobs *by* Drs. R. Theron and E. Krause.
Dr. J. O. Marais *by* Dr. A. W. Sichel, Dr. and Mrs. P. W. J. Keet, Dr. R. Theron, Mara and Clemens Keet.

Dr. R. E. Johnson *by* Dr. P. W. J. Keet.
Mrs. P. W. J. Keet *by* Dr. J. D. M. Classens, Dr. D. P. Marais.
Dr. M. Peskin *by* Dr. R. Schaffer, Drs. S. Heymann and S. N. Javett, Dr. A. L. Agranat, President and Members of Northern T.V.L. Branch.
Mrs. Gain *by* Dr. E. D. A. Orsmond.
Mrs. M. E. Bassett *by* Dr. M. Meyers.
Mr. J. C. Carter *by* Dr. A. H. Baxter.
Mr. F. A. C. Cuthrie *by* Dr. Vernon Brink.
Arie Dekker *by* Dr. M. Meyers.
Mrs. E. MacDornell *by* Dr. M. Meyers.
Mrs. E. Basson *by* Mr. and Mrs. P. v. Buddenbrock, Mr. A. K. Starke, Miss O. Starke, (Sister) D. S. van der Lith, Mr. and Mrs. J. Ovenstone, Linda de Villiers and Mary Maskew, David and Magda Versfeld, Mrs. E. F. Pretorius, Mrs. G. Morrison and Dr. and Mrs. N. C. Starke, Mrs. J. Bainlay.
Mrs. Munday *by* Dr. G. M. Malan.
Dr. J. D. Allen *by* Mr. D. G. P. Clifford.
Dr. P. A. Green *by* Dr. Guy Fehrsen, His Grandchildren.
Dr. D. J. Theron *by* M.A.S.A. Northern Transvaal Branch.
Mrs. H. R. Balcomb *by* Dr. M. Henderson.
Mrs. Grace Osborn *by* M.A.S.A. Natal Coastal Branch.
Total amount received from Votive Cards, £67 14s. 6d.

Services Rendered to:
Dr. A. J. Orenstein *by* Dr. E. Samuels.
Mrs. E. D. Fairbairn *by* Mr. M. B. Murless.
Dr. W. S. Alexander *by* Dr. Warner.
Manfred son of Dr. S. M. Green *by* Dr. W. Trubshaw.
Dr. J. D. Blackburn and Family *by* Drs. Ross and Airey.
Mrs. P. K. de Villiers *by* Dr. J. A. S. Marr.
Dr. A. S. Smith *by* Dr. S. Hoffman.
Dr. A. S. Smith *by* Dr. Monte Jackson.
Wife of Dr. A. Rosin *by* Sisters of St. Catherine's Home.
Dr. A. J. v. d. Spuy *by* Dr. J. L. v. Rhyn.
Dr. M. J. Goddefroy *by* His Colleagues.
Dr. M. D. Ingle *by* Mr. W. A. D. Trubshaw and Mr. J. N. Smith, Dr. F. W. Roberts and Dr. J. Gluckman.
Dr. and Mrs. G. Rosendorff *by* Drs. J. A. Cluver, Prof. J. de Villiers, A. D. Keet and J. D. Joubert.
Mrs. B. Kavalsky *by* Dr. J. Abelsohn.
Shirley Kavalsky *by* Drs. Haarberger and Goldin.
The Late Mr. W. F. Hallatt and Mrs. M. M. Hallatt *by* Drs. J. A. Hartman, J. D. Meyers, E. Hesselberg, von W. Eybers, G. Trichard.

Total amount received from Services Rendered, £279 19s. 0d.

Donations by:

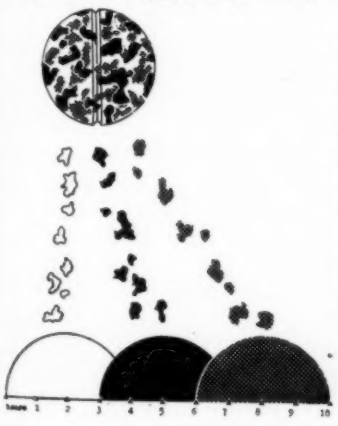
	£	s.	d.
Drs. Smith, Schaffer, Friedlander and Jacobson ..	5	0	0
Proceeds Annual Social Function E.R.B.	28	11	6
C.W. Branch members Collection Box	10	0	4
Certain Members N.C.B.	72	4	2
Members of Vaal River Branch ..	209	19	0
Drs. P. C. du Plessis and J. D. Verster	4	4	0
C.W.B. Members Collection Box ..	3	9	3
Proceeds of the Campbell Cup Golf Competition	11	10	0
Dr. M. J. Goddefroy	20	0	0
Dr. B. Epstein ..	10	0	0
Dr. P. R. Malherbe	0	10	0
Dr. W. R. Hackmann	0	10	6
Dr. A. C. L. Grantham	0	10	6
Dr. P. H. B. Maytom	0	10	6
Dr. A. A. Baxter ..	0	10	6
Dr. H. H. Broodryk	0	10	6
Dr. R. J. H. McMahon	0	10	6
Dr. P. A. Clarke ..	0	10	6
Dr. S. Disler ..	0	10	0
Dr. C. P. Bringle ..	1	1	0
Dr. A. J. P. Lessing	0	10	6
Dr. M. E. Brown ..	0	10	6
Dr. N. Shear ..	0	10	6
Dr. R. P. Seymour	0	10	6
Dr. E. T. Dietrich	1	1	0
Dr. E. E. le Roux ..	1	1	0
Dr. N. H. Helman	0	10	6
Dr. M. J. F. Davis	0	10	6
Dr. R. St. C. Sinclair	0	10	6
Dr. T. J. G. Haupt	0	10	6
Dr. A. L. Forbes ..	0	10	6
Dr. I. M. Hurwitz	0	10	0
Dr. T. S. Eddy ..	2	2	0
Dr. P. de V. Charlton-Perkins ..	0	10	6
Dr. D. M. Lithgow	0	10	6
Dr. J. C. W. Ehlers	0	10	6
Dr. H. L. Wallace	5	0	0
Dr. K. H. Dyke ..	0	10	6
Dr. M. J. Erasmus	0	10	6
Dr. G. P. Fourie ..	1	5	0
Dr. C. L. Wicht ..	0	10	6
Dr. J. H. Jackson ..	0	10	6
Dr. J. Smith ..	0	10	6
Dr. B. L. Butler ..	1	1	0
Dr. G. D. Morgan	1	1	0
Dr. J. Mibashan ..	0	5	0
Dr. R. T. Bokwe ..	0	7	6
Dr. J. S. M. Sililo ..	0	10	6
Dr. A. L. Rencken	0	10	6
Dr. G. Borok ..	2	2	0
Grand Total ..	£754	1	3

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

Belladenal Retard Tablets

Sandoz Limited, Basle, supply the following information:

Each Belladenal Retard Tablet contains 0·25 mg. of Bellafoline (total laevorotatory belladonna alkaloids equivalent in antispasmodic and anti-cholinergic effect to 27 minims of tincture belladonna) and 50 mg. (¾ gr.) of phenobarbitone.



The special feature of the Belladenal Retard Tablet is that the active ingredients are not all released at once, but over a period of 6 hours. One third (the white section) becomes effective immediately; the green particles become effective about 3 hours after ingestion, and the last third (orange particles) becomes effective after about 6 hours. Thus, the administration of one Belladenal Retard Tablet has a similar effect to the administration of 9 minims of tincture of belladonna and ¼ gr. of phenobarbitone at 3-hourly intervals, and provides adequate antispasmodic and anticholinergic effect for about 9-10 hours. This is of particular value in an evening dose, as it will provide an effective anti-cholinergic action throughout the night.

The bellafoline contained in Belladenal Retard Tablets consists of the total alkaloids of fresh belladonna leaves. In contrast to atropine, which is a racemic mixture of l-hyoscyamine with the inactive dextrotatory isomer, bellafoline contains only the natural laevorotatory forms. Consequently, it is almost twice as active as atropine, enabling the same therapeutic effect to be obtained with about half the dosage.

In Belladenal Retard Tablets the potent anti-cholinergic action of bellafoline is reinforced by the action of phenobarbitone on the autonomic centres in the brain stem. The mutual synergism between the two components ensures that a powerful anti-spasmodic and sedative effect is obtained with relatively small doses.

The indications for Belladenal Retard Tablets are: spasms of the gastro-intestinal and urinary tract; peptic ulcer; dysmenorrhoea; emotional tension; nocturnal enuresis, and all conditions where an anti-cholinergic effect is desired.

Presentation: Bottles of 20 and 100 Belladenal Retard Tablets.

Sole distributors: Alex. Lipworth Limited, P.O. Box 4461, Johannesburg, P.O. Box 1988, Durban and P.O. Box 4838, Cape Town. Samples and literature available on request from:

Alex. Lipworth Limited, Sandoz Pharmaceutical Department, P.O. Box 4461, Johannesburg.

INCOME TAX AS IT AFFECTS THE MEDICAL PRACTITIONER

As a result of negotiations instituted by Federal Council the Commissioner of Inland Revenue has intimated that subscriptions to the Medical Association and subscriptions to medical journals will rank as a deduction from the taxable income of doctors in full-time employment.

The Commissioner writes: 'After further consideration it has been decided to admit the claim and, with effect from the year of assessment ended 30 June 1957, Receivers of Revenue will, in respect of all medical practitioners in full-time employment, allow the actual cost of such subscriptions in the determination of the taxable income derived by them from their professional activities'.

Hitherto practitioners in private practice have been allowed this deduction: it is now extended to those in full-time employment.

PASSING EVENTS : IN DIE VERBYGAAN

Mr. R. C. J. Hill, F.R.C.S., of Durban, has been elected a Fellow of the British Orthopaedic Association.

* * *

Dr. Willem P. Steenkamp has returned to Cape Town from a visit to Denmark, where he has been attending surgical clinics in Copenhagen.

Dr. Willem P. Steenkamp het teruggekeer na Kaapstad van Denemarke, waar hy chirurgiese klinieke in Copenhagen bygewoon het.

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Dr. S. J. Edelman of Randfontein will be joining Dr. Harold Lewin of 10/11 Jenner Chambers, Jeppe Street, Johannesburg, in his practice as from 1 September 1957.

* * *

Dr. and Mrs. Merskey of Cape Town have returned from a trip to the U.S.A. Dr. Merskey left in December 1956 on a Rockefeller Travelling Fellowship in Haematology and has spent his time in the U.S.A., Italy and Great Britain.

* * *

The doctors of Vereeniging, through the Vaal River Branch of the Medical Association, have donated the sum of £209 19s. 8d. to the Benevolent Fund. This consists of remuneration which they recently received for assistance rendered at the Vereeniging Hospital.

* * *

Research Forum, University of Cape Town. The next meeting of the Research Forum will be held on Wednesday 7 August 1957 in the A-Floor Lecture Theatre, Groote Schuur Hospital, Cape Town, at 12 noon. Speaker: Dr. W. P. U. Jackson on behalf of the Diabetic Clinic. Subject: Oral hypoglycaemic agents in Diabetes—clinical trial.

* * *

The Royal Institute of Public Health and Hygiene conducts recognized Courses of Instruction annually (for postgraduate medical men and women only) for the Diploma in Public Health and the Diploma in Industrial Health of the Conjoint Board of the Royal College of Physicians of London and the Royal College of Surgeons of England, and for the Diploma in Industrial Health of the Society of Apothecaries of London. The next Courses of Instruction will commence on 20 September 1957. Further information, entry forms and prospectuses may be obtained from the Acting Dean at 23 Queen Square, London, W.C. 1, or from the Acting Secretary of the Institute at 28 Portland Place, London, W. 1.

* * *

A Course on the Therapies in Cerebral Palsy, organized by the Cerebral Palsy Division of the National Council for the Care of Cripples in South Africa, will be held on 30 September and 1-2 October 1957 at Medical House, Hospital Hill, Johannesburg. The course will consist of lectures, case demonstrations, films and an exhibition to give an introduction to the nature of cerebral palsy and its therapy. It will be divided into 3 portions, viz. 'Medical Background', 'The Therapies' and 'The Non-motor Handicaps in Cerebral Palsy'. A cerebral palsy school and treatment-centre in operation will be demonstrated.

The course is intended for therapists and professional personnel working with cerebral palsy cases. All interested medical practitioners are cordially invited to attend. Further details can be obtained from the Organizer, Miss S. Levitt, B.Sc. Physiotherapy (Rand), P.O. Box 10150, Johannesburg. Telephones 23-5496 and 23-5697.

* * *

South African Paediatric Association. The Cape Town Subgroup of this Association are holding a clinical evening at the Karl Bremer Hospital, Bellville, Cape, on Tuesday 6 August at 8.15 p.m. Cases will be shown from 7.45 p.m. All practitioners are welcome.

* * *

National Group of Neurologists, Psychiatrists and Neuro-surgeons (M.A.S.A.). A Symposium on *Cerebro-vascular Disease* will be held at Medical House, Esselen Street, Johannesburg on 7 August at 8 p.m. under the joint auspices of the Southern Transvaal sub-group of this National Group and the National Association of Physicians of South Africa. All practitioners are welcome.

* * *

University of the Witwatersrand Medical Graduates Association Alumni Dinner. A dinner at which the guests will be the Class of 1931-32 and their teachers and present members of staff has been arranged for Thursday 22 August at The Auto Club, Killarney, Johannesburg. Tickets £1 10s. 0d. (including drinks). Cocktails at 6.30 p.m. Dress formal. For reservations please apply to the Secretary, Medical Graduates Association, Medical School, Hospital Street, Johannesburg. Telephone 44-7040 in the forenoon from 9 to 12.30.

* * *

Unie van Suid-Afrika. Departement van Gesondheid. Aangifte van gedugte epidemiese siektes en poliomiëlitis in die Unie gedurende die tydperk 12 Julie—18 Julie 1957.

Poliomiëlitis

	Bl.	*Nat.*	*Kl.*	*As.*	*Totaal*
Transvaal	5	6	1	—	12
Kaapprovinsie ..	2	—	4	—	6
Oranje-Vrystaat ..	1	2	—	—	3
Natal	2	2	—	—	4
	—	—	—	—	—
Totaal ..	10	10	5	—	25

Pes, Pokkies, Tifuskoors: Geen.

* * *

Dr. H. J. Steyn of Windhoek has been elected to the Senate to represent South West Africa. The election took place at a meeting of the members of Parliament and the Legislative Assembly for South West Africa. Dr. Steyn was President of the South West Branch of the Medical Association of South Africa in 1953. He has been Mayor of Windhoek for the past 2 years.

REVIEWS OF BOOKS : BOEKRESENSIES

OBSTETRICS FOR THE GENERAL PRACTITIONER

Management in Obstetrics. Second Edition. By Andrew M. Claye, M.D., F.R.C.S., F.R.C.O.G. Pp. x + 218. 35 Figures. 18s. net. London: Oxford University Press. 1956.

Contents: Preface to the Second Edition. Preface to the First Edition. 1. Abortion. 2. Antenatal Supervision. 3. Breech Pregnancy. 4. Diet in Pregnancy. 5. Twins. 6. Excessive Vomiting. 7. Treatment of Retroversion with Pregnancy. 8. Hydramnios. 9. Fibroids in Pregnancy and Labour. 10. Hypertension and Pre-eclampsia. 11. Haemorrhage. 11*a*. Ante-Partum Haemorrhage. 12. Postmaturity. 13. Management of Labour. 14. Abnormal Uterine Action. 15. Relief of Pain in Labour. 16. Difficulty with Shoulders. 17. Occipito-Posterior Positions and Deep Transverse Arrest. 18. Breech in Labour. 19. Face and Brow Presentations. 20. Transverse Lie. 21. Presentation and Prolapse of the Cord. 22. Forceps (Indications and Conditions). 23. Obstetric Operations. 24. The Forceps Operation. 25. Failed Forceps. 26. Low Puncture of the Membranes. 27. Repair of Perineal Tears. 28. Episiotomy. 29. Retained Placenta. 30. Post-partum Haemorrhage. 31. Resuscitation. 32. Puerperium. 33. Retention of Urine after Delivery. 34. Breast-feeding. 35. Intramuscular Injection. 36. On Calling in Consultants. 37. Instructions on Masks. 38. Prevention of Puerperal Sepsis. Appendices. Index.

This little book by Professor Claye of Leeds has been written primarily for the general practitioner interested in midwifery and confines itself to problems of management. As the author indicates in his preface to the first edition (1948), it tends to be dogmatic, but it is not claimed that the lines of treatment described are above criticism.

The book is well written in a clear and concise style. The views expressed will be generally acceptable. The approach is always practical and the text is rich in sound words of obstetric wisdom. Because most of the chapters are reasonably short, general practitioners and final-year students will find this book the ideal one to dip into at odd times, and it will offer them wholesome and nourishing refreshment with welcome relief from their ordinary run of work and reading.

Another attractive feature is an introduction to most chapters with an apt quotation from English literature.

This 1956 edition contains new chapters on postmaturity and retained placenta, while many of the obstetric advances of the last few years have found a place in this excellent little handbook. I unhesitatingly recommend it to students in their final year and to those general practitioners who practise obstetrics.

E.M.S.

INTERNAL MEDICINE

Internal Medicine. A Physiologic and Clinical Approach to Disease. By Robert P. McCombs, B.S., M.D., F.A.C.P. Pp. xxi + 706. 49 Figures. $10.00. Chicago: Year Book Publishers, Inc. 1956.

Contents: 1. Disorders of the Heart. 2. Hypertension and Vascular Disease. 3. Renal Disease and Renal Failure. 4. Body Fluids and Electrolytes. 5. Endocrine Disorders. 6. Infectious Diseases. 7. Pulmonary Diseases. 8. Allergic Diseases. 9. Disorders of the Gastrointestinal Tract. 10. Disorders of the Blood and Blood Forming Organs. 11. Rheumatic Disorders and Diseases of Bone. 12. Nutritional and Metabolic Disorders. Index.

This is essentially a modern book. Brief lucid descriptions are given of the common disorders and diseases encountered in the practice of internal medicine. Stress is laid on the physiological principles and mechanisms underlying not only the clinical manifestations of disease but also the diagnostic methods and therapeutic measures advocated.

The fundamentals concerning the regulation of body fluids and electrolytes in health and disease are admirably reviewed and practical measures for the correction of aberrations are clearly defined. In clarifying a difficult though important facet of modern therapeutics, this chapter distinctly enhances the value of the volume. The same could be said of the chapter on renal disease and renal failure. Endocrine disorders are well presented, but the syndrome of primary aldosteronism is omitted. Infectious and pulmonary diseases are given detailed consideration. The therapeutic application of adrenocortical steroids and of anticoagulants is referred to freely throughout the volume. The section on diseases of the liver is distinctly inadequate; the mechanism and management of hepatic encephalopathy is omitted, hepatic coma is barely mentioned and ammonium chloride is still advocated as medication for liver cirrhosis. Elsewhere loose statements, omissions and inaccuracies can be found; for example, in the management of acute myocardial infarction, quinidine administered by the intravenous route is recommended for the *prophylaxis* of ventricular tachycardia, and the well-proven value of intravenous infusion of norepinephrine for the control of shock receives no mention. In figure 46, it is stated that parathyroid hormone inhibits excretion of phosphate by the kidney. On the assumption that neurology and psychiatry are independent specialities these subjects are excluded from the volume, and this is regretted, but the same justification cannot be claimed for the scant, vague and often contradictory references made to psychosomatic disorders and anxiety, the recognition and management of which constitute an important function of the modern internist. By and large, however, this is a useful volume to possess; it brings together, in a concise readable form, many of the essentials of modern internal medicine.

M.M.S.

BOERHAAVE SE BRIEWE AAN BASSAND

Boerhaave's Brieven aan Bassand. Vertaald en geannoteerd door Prof. Dr. G. A. Lindeboom. Pp. xvi + 273. f 15. Haarlem: De Erven F. Bohn N.V. 1957.

Inhoud: Voorwoord. Joannes Baptista Bassand. Boerhaave's Brieven aan Bassand. Bericht van Zijn Overlijden aan Bassand. Enige Jaartallen.

Hermann Boerhaave (1668-1738), op sy dag stellig die beroemdste geneesheer in Europa, het in Leiden studeer. In 1684 begin hy sy studies in die godgeleerdheid en Oosterse tale en in 1690 promoveer hy tot doktor in die wysbegeerte. Vervolgens gaan hy oor tot die geneeskunde en in 1693 promoveer hy tot dokter in die medisyne. In 1701 word hy aangestel as lektor in teoretiese geneeskunde te Leiden en in 1709 word hy bevorder tot hoogleraar in plantkunde en geneeskunde aan dieselfde Universiteit. Sy twee grootste werke, ‚Institutiones Medicae' (1708) en 'Aphorismi de Cognoscendis et Curandis Morbis' (1709), word in verskeie Europese tale sowel as Arabies vertaal. In 1718 neem hierdie merkwaardige en vermaarde geleerde ook die professoraat in skeikunde op hom.

Johannes Baptista Bassand (1680-1742) is in die graafskap Boergondië in Frankryk gebore. Hy lê hom toe op die studie van snykunde in Parys en Napels maar gaan oor tot die geneeskunde in Salerno, vanwaar hy na Leiden verhuis om by Boerhaave sy studies voort te sit. Hier ontwikkel daar 'n hegte vriendskap tussen leraar en leerling. Bassand word later opperste chirurg in die Oostenrykse leër in Italië en in 1712 die lyfarts van die hertog van Savoje in Weenen. Hy maak snelle vordering en word in 1728 tot die adelstand verhef, terwyl hy die volgende jaar tot lyfarts van die Keiserlike gesin benoem word. Kort daarna kry hy die titel ‚Baron'. Bassand sterf in 1742.

Vanaf 1714 tot sy dood in 1738 het Boerhaave 'n briefwisseling (in Latyn) met Bassand gevoer. Laasgenoemde het byna al die briewe van sy eertydse leermeester bewaar, maar ongelukkig ontbreek die briewe van Bassand aan Boerhaave. Hierdie briewe verskyn nou vir die eerste keer in 'n Nederlandse vertaling, keurig versamel deur Professor Lindeboom. Die boek is ook deeglik voorsien van leersame en inligtingryke voetnote. Die briewe bevat grotendeels antwoorde op vrae van Bassand in verband met een of ander siektegeval. Maar daar is ook briewe oor edelgesteentes, kwiksilwer, skeikundige proewe, metale, plantkunde, geneeskundige kruie en dies meer.

Hierdie interessante werk, mooi ingebind en versorg, sal aan menige praktisyn genotvolle ontspanning vir 'n ledige uurtjie verskaf.

E.M.S.

SKIN TUMOURS

Die Geschwülste der Haut. Ihr Klinisches und Feingewebliches Bild, Ihre Erkennung und Behandlung. Von Prof. Dr. Dr. A. Greither und Dr. H. Tritsch. x + 280 Seiten. 180 Abbildungen. DM 49.50. Stuttgart: Georg Thieme Verlag. 1957.

Inhaltsverzeichnis: Vorwort. Einleitung. *A. Nicht-echte Geschwülste des Ektoderms.* I. Erregerbedingte reaktive geschwulstartige Bildungen. II. Reaktive geschwulstartige Bildungen durch chemisch-physikalische Reize. III. Das Rhinophym. *B. Nicht-echt Geschwülste des Mesoderms.* IV. Reaktive geschwulstartige Bildungen des Bindegewebes. V. Umschriebene geschwulstartige Bildungen durch Stoffwechselkrankheiten. VI. Reaktive granulomatöse, geschwulstartige

Bildungen. VII. Systematisierte Lipoid-Speicherkrankheiten. *C. Echte Geschwülste des Ektoderms.* VIII. Die ektodermalen Naevi. IX. Naevoide Basaliome. X. Basaliome. XI. Cysten. XII. Mischtumoren. XIII. Die sog. Pseudocancerosen. XIV. Die sog. Präcancerosen. XV. Der Stachelzellkrebs. XVI. Carcinome der Hautanhangsgebilde. XVII. Die malignen Melanome. *D. Echte Geschwülste des Mesoderms.* XVIII. Mesodermale Naevi. XIX. Gefässmäler und gutartige Gefässgeschwülste. XX. Gutartige Geschwülste nach Art des differenzierten Bindegewebes. XXI. Bösartige Geschwülste nach Art des differenzierten Bindegewebes. XXII. Gutartige Geschwülste nach Art des undifferenzierten Bindegewebes. XXIII. Bösartige Geschwülste nach Art des undifferenzierten Bindegewebes. XXIV. Die bösartigen Granulomatosen. *E. In die Haut abgesiedelte Geschwülste.* XXV. Absiedlung ektodermaler Geschwülste in die Haut. XXVI. In die Haut abgesiedelte mesodermale Tumoren.

The tumours here described are largely the blastomas, with short excursions outside this field to some inflammatory and hyperplastic lesions and the metabolic diseases. Clinical and histological features, differential diagnosis and treatment are shortly but adequately described and key references to the recent literature are given. The most attractive feature of the book is the illustrations; both clinical photographs and photomicrographs are, without exception, of the highest quality.

The compass of the work is large and most of the rarities are noted, but there is only fleeting reference to the mastocytoses and no mention at all of the concept of sarcoidosis as a reticulosis. A short account of the chronic granulomas and sarcoidosis from the point of view of differential diagnosis would, we feel, have been more relevant than some of the material included as, for example, Peyronie's disease, Dupuytren's contracture and the nodular lesions of acrodermatitis atrophicans.

This is a useful and trustworthy reference book.

J.M.

BRAIN ABSCESS

Der Hirnabszess. Von Gerhard Weber. viii + 185 Seiten. DM 29.70. Stuttgart: Georg Thieme Verlag. 1957.

Inhalt: Vorwort. *I. Einleitung. II. Allgemeine Pathogenese und Ätiologie.* A. Zur Pathogenese der traumatischen intrakraniellen pyogenen Infektion. B. Die Pathogenese der im Gefolge von Infektionen am Kopf auftretenden, der rhinogenen und der otogenen intrakraniellen entzündlichen Komplikationen. C. Die hämatogen-metastatischen intrakraniellen pyogenen Prozesse. *III. Symptomatologie und Therapie Intrakranieller Eitriger Infektionen.* A. Osteomyelitis des Schädels, Pachymeningitis externa und extraduraler Abszess. Klinik und Symptomatologie. B. Thrombosen intrakranieller Sinus und zerebraler Venen. C. Infektionen des Subduralraumes. Subdurale Empyeme und Abszesse. D. Die eitrigen Leptomeningitiden. E. Die Hirnabszesse.

This book, although entitled 'The Brain Abscess', deals with all the intracranial infections. The first part deals with the general pathology and etiology and is very thorough. The second part deals with the symptomatology and therapy of the intracranial infective processes.

The importance of performing air studies or angiograms, on patients who show any symptoms which might be due to a brain abscess, is stressed, as this is often the only way to reach a definite diagnosis.

The various forms of treatment, from simple aspiration to radical removal of the abscess, are dealt with in detail.

Although it is difficult to judge from statistics which is the best method of treatment, it would seem that, especially in the chronic brain abscess, radical removal gives the best results. Stress is laid on the necessity of adequate antibiotic treatment as an additional measure, whatever form of surgical treatment is employed.

The book gives a very good general concept of the modern treatment of intracranial infective processes, and should be read by all doctors, whether the subject has anything to do with their specialty or not.

H.L.deV.H.

PAEDIATRICS FOR NURSES

The Essentials of Paediatrics for Nurses. By I. Kessel, M.B., B.Ch., M.R.C.P. (Lond.), M.R.C.P. (Edin.), D.C.H. (Eng.). Pp. xii + 213. 88 Figures. 5 Colour Plates. 21s. net + 11d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1957.

Contents: 1. The Normal Child. 2. Prematurity. 3. Breast Feeding. 4. Artificial Feeding. 5. Affections of the Newborn. 6. Disorders of the Alimentary System. 7. Diseases of the Respiratory System. 8. Tuberculosis. 9. Disorders of Nutrition. 10. Disorders of the Blood. 11. Juvenile Rheumatism. 12. Disorders of the Cardiovascular System. 13. Disorders of Metabolism. 14. Diseases of the Genito-Urinary System. 15. Diseases of the Nervous System. 16. Functional Nervous Disorders. 17. Mental Deficiency. 18. Diseases of the Endocrine System. 19. Some Surgical and Orthopaedic Conditions of Infancy and Childhood. 20. Congenital Syphilis. 21. Diseases of the Skin. 22. Acute Infections and Contagious Fevers. 23. Common Poisonings in Childhood. 24. Common Paediatric Procedures. *Appendix:* I. Weights and Measures and Dosage Equivalents. II. Useful Information. III. Questions Set at the Final State Examination for the Part of the Register for Sick Children's Nurses. IV. Some Questions set for the Examination for the Certificate in Paediatric Nursing of the South African Nursing Council. V. A Short Account of the Antibiotics and Sulphonamides. Index.

This small book is dedicated to the nursing profession and the reviewer thought that an auspicious opening. But the farther he read the more he became convinced that the book was unsuitable for most nurses. This is very disappointing, for it is obvious that the author started his work with a good idea in his mind and allowed it to wander off into a technical wilderness which is not even connected by a side-road with the highways trodden by nurses.

On almost every page there appears some information incomprehensible to and irrelevant for paediatric or any other nurses; 'This sensitization produces the pathological lesion of an Aschoff's node, which is seen microscopically to be made up of giant cells and fibroblasts.' 'Thromboplastinogen, which interacts with the thromboplastinogenase of the platelets to form thromboplastin.' Under no concatenation of circumstances can such items be regarded as part of the essentials of nursing knowledge, which should include sufficient information to enable the nurse to understand why and when she must do this or that. She is not told that.

An immense amount of work has gone astray. A pity.

F.J.F.

FOR ADVANCED STUDENTS IN SURGERY

Companion in Surgical Studies. Second Edition. By Ian Aird, Ch.M., F.R.C.S. and The Staff of the Department of Surgery, Post graduate Medical School. Pp. xii + 1302. 84s. net. Edinburgh and London: E. & S. Livingstone Ltd. 1957.

Contents: I. Introduction. II. The Local Effects of Trauma. III. The General Effects of Injury and Haemorrhage. IV. Blood Transfusion. V. Water and Electrolyte Replacement Therapy; Intravenous Alimentation. VI. Pyogenic Infection. Chemotherapy. VII. Specific Infections. VIII. Tumours. IX. The Arteries. X. The Veins. XI. The Lymphatic Vessels, Lymph Nodes and Reticular Tissues. XII. The Mouth. XIII. The Tongue. XIV. The Jaws. XV. The Salivary Glands. XVI. The Skull. XVII. The Cranial Contents. XVIII. The Spine and Spinal Cord. XIX. The Peripheral Nerves. XX. The Sympathetic Nervous System. XXI. The Neck. XXII. The Thyroid Gland. XXIII. The Parathyroid Glands. XXIV. The Larynx, Trachea and Pharynx. XXV. The Oesophagus. XXVI. The Chest Wall. XXVII. The Lungs and Pleura. XXVIII. The Heart and Pericardium. XXIX. The Mediastinum. XXX. The Breast. XXXI. The Abdominal Wall. Abdominal Injuries. Hernia. XXXII. The Peritoneal Cavity, Mesentery and Omentum. XXXIII. The Stomach. XXXIV. The Duodenum. XXXV. Intestinal Obstruction. XXXVI. The Small Intestine. XXXVIII. The Colon. XXXIX. The Rectum and Anus. XL. The Liver, Gall-bladder and Bile Ducts. XLI. The Pancreas. XLII. The Spleen. XLIII. The Adrenal Gland. XLIV. The Kidney and Ureter. XLV. The Bladder. XLVI. The Prostate. XLVII. The Urethra and Penis. XLVIII. The Scrotum, Testis, Epididymis and Spermatic Cord. XLIX. Amputations. Appendices. Index.

This book first appeared in 1949, and durng the relatively short period since that time it has become the foiremost reference work in the English language for advanced students of surgery. This is probably as high a recommendation as any work of this kind can earn, since these students are much more thorough and critical than any reviewer is likely to be.

The work is of course encyclopaedic in its compass, and not the least of its merits lies in the truly remarkable index system which enables one to locate almost any surgical topic in a few seconds. As in the previous addition the bibliography is most handily placed at the foot of the page on which the subject appears. The latest edition contains considerable new material and the inclusion of recent advances brings it up to the minute. The following are among the new and revised subjects:

The present improved treatment of actinomycosis and of carcinoma of the tongue is revised and better reasoned than previously. The section on the thyroid gland has been widely re-written to include modern data on radio-active iodine. This has modified the approach to diagnosis and treatment in both toxic and malignant disease of the thyroid. The treatment of cardiospasm has been improved to exclude cardioplasty and other operations likely to be followed by oesophageal reflux. The section on diaphragmatic hernia—at present a surgically fashionable disease—has a revised classification but the revision has not been extended to include treatment. The revision of the therapy of carcinoma of the breast brings this section up to date by including not only a discussion on radio-therapy but also recent work on hormone therapy, adrenalectomy etc.

The latest edition maintains the place of this book in the forefront of reliable works on the subject and it earns as high a recommendation in 1957 as the first edition did in 1949.

P.C.W.M.

MEDICAL DICTIONARY

Dorland's Illustrated Medical Dictionary. Twenty-third Edition. Editorial Board: Leslie Brainerd Arey, Ph.D., Sc.D., LL.D., William Burrows, Ph.D., J. P. Greenhill, M.D., and Richard M. Hewitt, A.M., M.D. Pp. xvii + 1598. More than 700 illustrations and 50 plates. $12.50. Philadelphia and London: W. B. Saunders Company. 1957.

Contents: Consultants and Major Contributors. Preface. Notes on Use of This Dictionary: Arrangement of Entries, Alphabetization, Pronunciation, Plurals, Etymology, Abbreviations Used in Definitions. Index to Plates. Dictionary of Medical Terms: Naturally-Occurring Amino Acids, Chief Malaria-Carrying Anopheles Species of the World, Arteries, Chemical Elements, Gram-Negative and Gram-Positive Bacteria, Endocardial Murmurs, Muscles, Nerves, Positions of the Child in Utero in Various Presentations, Important Poisonous Snakes of the World, Stains and Staining Methods, Tests, Centigrade and Fahrenheit Thermometric Equivalents, Tables of Weights and Measures, Metric Doses with Approximate Apothecary Equivalents. Modern Drugs and Dosage. Fundamentals of Medical Etymology.

The 23rd edition of this reference work initiates a new era of its existence. The original motivating policy of producing, 'in a volume of convenient size, an up-to-date medical dictionary, sufficiently full for the varied requirements of all classes of medical men', remains unchanged, but the book has been rechristened *Dorland's Illustrated Medical Dictionary*, in conformity with the appellation by which it has become familiarly known to the generations of physicians who have used it'.

This fantastically useful book has now entered its 23rd edition. Its popularity is well deserved and it forms the most reliable guide to English medical terminology.

The print is clear, the paper excellent and the binding strong and handy. It is a credit to the publishers and is without doubt the finest reference book available for the medical editor.

The differences that exist between American and English medical usage are unfortunately not brought out. It remains for an English firm to bring out a dictionary of similar excellence to fill the gap. We may well not see this task fulfilled in our time.

W. A. Newman Dorland, A.M., M.D., F.A.C.S., whose name as editor appeared on the title page of the American Illustrated Medical Dictionary, as the book was called, in each of the 22 preceding editions, died in September 1956, at the age of 92, and his name is now enshrined in the new title of his masterpiece. Dr. E. C. L. Miller who, as professor of Bacteriology and Biochemistry at the Medical College of Virginia, in 1923 became a significant contributor to the dictionary, died in July 1954, at the age of 87. The groundwork established by these men in the past becomes the solid foundation upon which the future achievements of the Dictionary will rest.

T.S.

THE EYE IN GENERAL PRACTICE

The Eye in General Practice. By C. R. S. Jackson, M.A., B.M., B.Ch. (Oxon.), D.O.M.S., F.R.C.S. (Ed.). Pp. vii + 152. 25 Figures. 21s. net. + 11d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1957.

Contents. Part I. Diseases of the Eye. I. Examination of the Eye. II. The Eyelids. III. The Conjunctiva. IV. The Watery Eye. V. The Cornea. VI. Intraocular Inflammation. VII. Cataract. VIII. Diseases of the Retina. IX. Errors of Retraction and Ocular Headache. X. Squint. XI. Glaucoma. XII. Injuries to the Eye. *Part II. The Eye in Diseases of Other Parts.* XIII. The Eye in Cardiovascular Disease. XIV. The Eye in Intracranial Disease. XV. The Eye in Endocrine and Metabolic Disease. XVI. The Eye in Diseases of Anatomically Related Parts. XVII. The Eye in Other Conditions. *Part III. Administrative.* XVIII. The Welfare of the Blind. XIX. The Ophthalmic Side of the National Health Service. Index.

In the short period allotted to the medical student's training in ophthalmology, the problem is how best to summarize the subject, what to omit and what to stress. For most students, the result is that they remember acute glaucoma, and the man with the enlarged liver and the glass eye. The subject is otherwise considered too recondite, and its practice is subsequently ineffectually pursued.

This short book, whilst not parading as a systematic text-book, will to some extent remedy this defect. It is a simplified, concise description of the common eye conditions, written almost for the lay reader. It suffers, therefore, from being rather dogmatic, and the substitution of the word 'usually' for 'invariably' in statements like 'purulent conjunctivitis in adults is invariably gonorrhoeal' will not weaken the sense. There are several controversial points in treatment, such as that atropin is advised in hyphaema, and that radiotherapy is preferred to surgery in rodent ulcers of the lid.

Nevertheless, this book should prove extremely useful to the practitioner and student as an aid in recognizing the common conditions, in analysing consultants' reports, and in reassuring and explaining the patient's condition where the consultant has failed to be sufficiently clear. In this latter respect, the chapters on squint and ocular headaches are particularly helpful. Some useful points chosen at random are the following: In wounds of the lids, excision of the wound edges and of badly torn tissue is not advisable. In lime burns, irrigation with saturated glucose solution converts the residual lime to insoluble calcium gluconate. In treating conjunctivitis, the most important measure is to avoid covering the eye, especially with the pernicious celluloid eye-shield.

L.S.

PURINES

Ciba Foundation Symposium on the Chemistry and Biology of Purines. Editors for the Ciba Foundation G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch. and Cecilia M. O'Connor, B.Sc. Pp. xii + 327. 124 Illustrations and structural formulae. 48s. net. London: J. & A. Churchill Ltd. 1957.

Contents: Opening remarks. Synthesis and properties of purines of potential biological interest. Some synthetic studies on purines and related heterocycles. Some new N-methylpurines. The structure of the hydroxypurines investigated by O- and N-methylation. The spectra and structure of the monohydroxypurines and other potentially tautomeric purines. The π-electron properties of purine calculated by the L.C.A.O. method. The degradation of uric acid by water under pressure. The 8-position in purines. The chemical and biological transformation of purines into pteridines. Cyclonucleosides. Stereochemistry of nucleoside synthesis. Synthetic chemical investigations related to the metabolism of purines. The effects of potential antipurines on a purine-requiring strain of *Escherichia coli*. The chemistry of new purines in the B_{12} series of vitamins. Biological and microbiological activity of purine analogues of vitamin B_{12}. Puromycin. Chemical and biological behaviours of 9-β-D-ribofuranosylpurine. On the activation of the one-carbon unit for the biosynthesis of purine nucleotides. The enzymatic synthesis of inosinic acid *de novo*. Enzymic control of purines by xanthine oxidase. The biological effects of 8-azapurines. Biochemical effects of 6-mercaptopurine. The use of 6-mercaptopurine in the treatment of leukaemia. General Discussion. Chairman's closing remarks.

This book is an account of a Ciba Foundation symposium on the chemistry and biology of purines which was held in May 1956. The conference was chiefly concerned with the synthesis of purine derivatives and their biological effects. The book records over 20 of the papers presented and the discussions which followed.

The purines form an essential part of the structure of nucleic acids, and this has stimulated searches for modifications and substitutions which it is hoped will provide new agents for modifying nuclear structure and for the chemotherapy of infections and tumour growth.

This report of progress along these lines will be of great value and interest to chemists and biologists familiar with its background; this is its purpose. It is highly specialized and very little of it will be helpful to those without a fairly good knowledge of this field.

G.C.L.

Biology of the Treponematoses, by Thomas Bourne Turner and David H. Hollander, Geneva, 1957 (*World Health Organization: Monograph Series*, No. 35), 278 pages. Price: £1 10s., $6.00, or Sw.fr. 18. French and Spanish editions in preparation. Local Sales Agent: Van Schaik's Bookstore (Pty.), Ltd., P.O. Box 724, Pretoria.

This monograph is based on investigations carried out at the International Treponematosis Laboratory Centre in the Department of Microbiology of the Johns Hopkins University. It is arranged in 3 parts. Part I deals with the biology of treponemal infections and includes chapters on the sources of strains used during the investigations, the experimental disease in laboratory animals, factors affecting its evolution, characteristics of treponemes *in vitro*, immunity phenomena, and the response of treponemes to drugs. Part II, which comprises a comparative study of treponemes, discusses the characteristics of the experimental disease invoked by various strains isolated from different parts of the world, the immunological relationships between these strains; and their behaviour in response to penicillin. Part III consists of a single chapter providing a useful summary of the principal findings set forth in preceding sections.

PAEDIATRICS

Advances in Paediatrics. Volume IX. Edited by S. Z. Levine. Associate Editors: John A. Anderson, Margaret Dann, A. Ashley Weech, Myron E. Wegman and Warren E. Wheeler. Pp. 336. Illustrations. $9.00. Chicago: Year Book Publishers, Inc. 1957.

Contents: Postmaturity by Stewart H. Clifford. The Gamma Globulins by Charles A. Janeway and David Gitlin. Thyroid Disorders in Childhood by William A. Reilly. Familial Dysautonomia by Conrad M. Riley. Use of Fluoride Compounds in Prevention of Dental Caries by Edward R. Schlesinger and David B. Ast. Coagulation Disorders in Infancy and Childhood by Irving Schulman and Carl H. Smith. Celiac Disease by H. A. Weijers, J. H. Van De Kamer and W. K. Dicke.

Designed for the paediatrician, this book is concerned mainly with problems that the general practitioner encounters in his everyday practice.

Postmaturity, which ranks so high as a cause of foetal and neonatal disease and death, was long overdue for such a comprehensive and authoritative review.

The gamma globulins function as antibodies. Their measurement has become a routine measure in laboratories, and practitioners should be familiar with the clinical conditions denominated as a-gammaglobulinaemia and hypo- and hypergammaglobulinaemia. In this admirable review more space could perhaps have been devoted to these clinical conditions.

Thyroid disorders are concisely covered, as is the difficult subject of coagulation disorders of the blood. There has been a vast increase in knowledge of the normal coagulation mechanism and this survey will give the paediatrician a working concept of this mechanism and thus lead to a more intelligent ordering of laboratory tests.

Familial dysautonomia is a new and unfamiliar term to describe a syndrome most often seen in children of Jewish extraction. The infant, who is a difficult feeder, has absent deep tendon reflexes. Later he cries, but does not weep, is hypotonic, perspires freely and has cold hands and feet. If in addition there is poor motor coordination and emotional instability—familial dysautonomia is the answer. It is an interesting study of a new and somewhat rare symptom complex.

Dental caries is a medical as well as a dental problem. Water fluoridation or even topical application of fluoride solution affords considerable protection against caries, and apparently, the only contra-indications to the use of fluorine are emotional arguments. This article is a challenge to all responsible medical practitioners.

To end this book is a chapter on coeliac disease. The relationship of the steatorrhoea to wheat gluten (gliadin) is clearly shown, but the essential abnormality remains unknown and coeliac disease is still a diagnosis by exclusion from coeliac syndrome.

All in all an excellent book for paediatrician and general practitioner.

S.C.S.

CORRESPONDENCE : BRIEWERUBRIEK

FARMASEUTIESE INRIGTINGS

Aan die Redakteur: U redaksionele opmerkings[1] in verband met finansiële ondersteuning wat deur die groter farmakologiese inrigtings aan geneeskundige navorsing bestee word, vind algemene byval. Dit is sekerlik nodig om hierdie feite onder die aandag van u lesers te bring.

Op 'n vroeë stadium in ons Departement word daar reeds ruim ondersteuning ondervind vanaf 'n Switserse maatskappy. Die fondse word beskikbaar gestel sonder voorwaardes.

Ek ag dit nodig om te benadruk dat dit ook nie die eerste geleentheid is waarby ek gou—en spontaan—hulp aangebied word deur Kontinentale firmas nie.

Hopelik sal daar meer dikwels in die toekoms deur hierdie firmas, ook deur Amerikaanse firmas, fondse beskikbaar gestel word vir plaaslike navorsing, eerder as vir buitelandse studie en werk.

Andries J. Brink

Karl Bremer-Hospitaal, Bellville, Kaap — *Professor van Interne Geneeskunde, Universiteit van Stellenbosch*

22 Julie 1957

1. Van die Redaksie (1957): S. Afr. T. Geneesk., **31**, 635.

WOODSTOCK TRAIN DISASTER

The following letter was addressed to the Secretary, Medical Association of South Africa.

Dear Sir: I desire to place on record the Administration's grateful appreciation of the spontaneous and willing manner in which the wireless appeal for medical assistance at the scene of the unfortunate collision between two passenger trains near Cape Town on 8 April 1957 was responded to.

It is most gratifying to learn that, because the appeal was answered so promptly, medical assistance was available almost immediately and the passengers in need of urgent attention were attended to with a minimum of delay.

I wish, therefore, to take this opportunity of expressing the thanks of this Administration, as well as my personal appreciation for the valuable assistance rendered by members of your Association in this instance.

D. H. C. du Plessis
General Manager

South African Railways
Johannesburg

29 June 1957

THE THREAT OF HYDATID DISEASE TO THE SOUTH AFRICAN CITIZEN

To the Editor: In the *Journal* of 13 July 1957 there appeared an article on this subject by Dr. I. F. de Villiers.[1] The warning it contained comes none too soon, and it would be well if it were heeded.

It is always a difficult matter to awaken the public to a consciousness of a danger when there has been no personal experience of it which has had a direct emotional impact. Nevertheless, repeatedly bringing a matter to the attention of the public has some effect, if it is vividly and forcefully hammered home.

Dr. de Villiers points the way to combating the spread of infestation by *Taenia echinococcus*, but it seems to me doubtful that his words will evoke a determined and public-spirited campaign to eradicate this tapeworm unless the public-health authorities take the matter up energetically. Certainly Dr. Alexander's advice (which he quotes) to refrain from fondling and caressing dogs is unlikely to be followed by the many thousands of people who number at least one dog in their households.

In New Zealand, where I was living recently, the authorities and the public are now alive to the menace of hydatid disease in their midst; and I should suggest that their methods of combating this worm should be adopted in the Union of South Africa. The New Zealand local authorities issue a warning and explanatory circular with the annual dog-licence reminder. When the licence is issued, worm capsules (of adequate strength) are issued *gratis* with the licence. This means that every licensed dog is wormed for hydatid at least once a year. Further, from time to time, the authorities place conspicuous and eye-catching advertisements in the daily press emphasizing the dangers of hydatid disease, and urging the public not to give offal to their dogs as food, and to see that all meat for dogs is cooked and that dogs are wormed regularly. It may be that the cooking of dog meat is not necessary here, but in New Zealand the staple meat is mutton, and much of it is infested.

In my own home I have an enclosed kitchen garden with 6-foot pig-netting round it, but I notice that very many people grow lettuces etc. where dogs have easy access to them, a habit to be deplored.

C. Holmes

9 Melrose Avenue
Westville
Natal

1. de Villiers, I. F. (1957): S. Afr. Med. J., **31**, 700.